BLOOD-PRESSURE

IN

MEDICINE AND SURGERY

A GUIDE FOR STUDENTS AND PRACTITIONERS

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TO

MY WIFE

PREFACE.

In the opinion of so eminent a physiologist as Ludwig, the discovery of blood-pressure by Stephen Hales was more important than that of the circulation of the blood by Harvey. As far as its clinical application was concerned, the study of blood-pressure languished for many years, owing to the difficulty which attended its estimation in man. With the description by Riva-Rocci, in 1899, of an instrument which, by its very simplicity, immediately won universal recognition, the importance of sphygmomanometry began to be appreciated.

The clinical study of blood-pressure is but a decade old, but in these few years it has furnished a great amount of diagnostic, prognostic, and therapeutic information. Some there are who still cling to the belief that the finger is just as trustworthy in estimating the arterial pressure as is the sphygmomanometer, but to those who have given the subject more careful consideration this contention seems extravagant. The sense of touch is, in some, more highly developed than in others; yet the man who seriously states that he is able to measure bodily temperature by palpation alone, is no more in error than is he who presumes to gauge the blood-pressure by means of the finger. An argument for the truth of this statement may be afforded to anyone who will palpate the radial artery and "guess," for it is only a conjecture, the degree of blood-pressure before using

the sphygmomanometer. The marked discrepancies between the results furnished by the two procedures, even after many years in practice, will be proof enough of the value of the instrument. The sphygmomanometer is now as much a part of the physician's proper armamentarium as are the clinical thermometer and the stethoscope, and the assistance which may be expected from its routine employment should also be as familiar to him.

It has been the author's aim to make fully available the assistance which the study of blood-pressure affords in the diagnosis, prognosis, and treatment of disease. The essentials are covered, and if after mastering them the reader is so disposed, he may range at will in an almost inexhaustible literature. E. H. G.

Philadelphia, 1914.

CONTENTS.

BLOOD-PRESSURE.

CHAPTER I.

PHYSIOLOGY OF BLOOD-PRESSURE.

It is essential, in studying blood-pressure, that one should be informed concerning its physiology, for without knowledge of the physiological mechanism which plays such an important role, an intelligent comprehension of the sphygmomanometer is impossible. The human blood-pressure system comprises essentially, (1) the heart; (2) the bloodvessels; and (3) the vasomotor regulating mechanism. The three are so intimately associated that disturbance of any one is followed by a derangement of the balance existing among the three.

I. MAINTENANCE OF BLOOD-PRESSURE.

Heart and Bloodvessels.—As far as the heart and bloodvessels are concerned, blood-pressure depends essentially on three factors: (*a*) the energy of the heart; (*b*) the peripheral resistance, and (*c*) the volume of the blood. The first two are of great importance, while the last has but a theoretical interest.

II. BLOOD-FLOW IN ARTERIES.

The vascular tree consists of a finely divided system of elastic tubes, the blood flowing through the arteries, through the capillaries, to the veins, the pressure being highest in

the arteries and lowest in the veins. This is designed for the movement of the column of blood, as a fluid must flow from the point of highest pressure to the point of lowest pressure. It is through the activity of the heart that the difference in pressure is maintained. When, for any reason, the heart ceases to functionate, the circulation continues, that is, the blood flows, despite the failure of the heart, from artery to vein, until the pressure in these two branches of the vascular system has become equalized, at which moment the blood stream comes to a standstill.

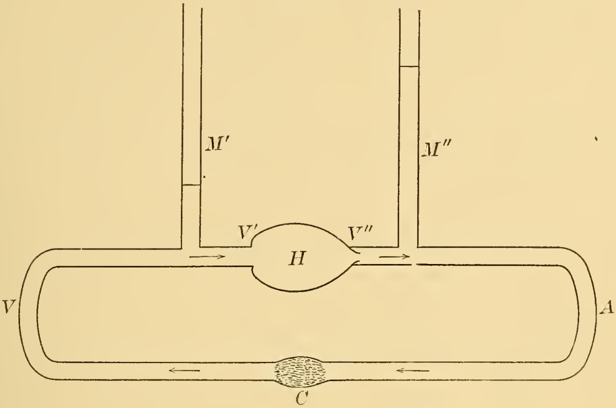
Despite the periodic activity of the heart, the blood is sent through the vessels in a continuous current, the reasons being, (1) the resistance offered by the arterioles and capillaries, and (2) the elasticity of the arterial wall. These two physicial properties of the vessels prevent stagnation of the blood during diastole, and account for its propulsion during this phase of the cardiac cycle. The elastic property of the arteries is especially concerned, for the vessel being put on the stretch during systole, stores up this energy to discharge it during diastole, thereby contracting the artery and expelling the blood. One might almost speak of this as an echo of the cardiac systole arising in the arteries.

Langendorff's modification of Weber's scheme of the circulation, conveys a good idea of the general physical features of the latter.

H is a rubber bag corresponding to the heart, V' and V'' represent the inlet and outlet valves of the heart. The rubber tubing A is the arterial and V the venous system, each being provided with a pressure gauge, M'' and M' respectively. C is a sponge in a glass tube which represents the resistance offered by the capillaries, the whole apparatus being filled with water. If one presses rhythmically on the bulb H, the valves V' and V'' act as *in vivo*, the column of water rises in M'' and sinks in M', while the tube A becomes more tense and the tube V more relaxed. As a result of the cardiac activity (H) the pressure in the arteries is higher than in the veins, owing to the resistance in C. In the diastolic pause (release of pressure on the bulb) the pressure in A falls, and that in V rises, and the water is sent forward

despite the cessation of the activity of *H*. If particles are suspended in the water, the movement of the fluid can be plainly seen.

FIG. 1



Weber's scheme of the circulation.

When compression on *H* is discontinued, the circulation persists until the column of fluid in *M″* stands at the same level as that in *M′*. This experiment serves to confirm the statement that the principal driving force for the arterial circulation is to be found in the heart and elasticity of the vessel walls. In addition to this force may be mentioned the contractile power of the arteries inherent in the smooth muscle fibers of the media.

III. BLOOD-FLOW IN CAPILLARIES AND VEINS.

By the time the blood arrives at the smaller arteries, little is to be seen of the rhythmic activity of the heart, that is, pulsation is generally no longer appreciated, and when the capillaries are reached, there is no pulsation at all (excepting in pathological states), although they do possess the power of contraction. By the time the blood gets to the veins, the force of the cardiac systole is *nil,* and

for the propulsion through this part of the vascular tree, the negative pressure in the thorax and the aspiration of blood by the diastole of the heart are called into play.

Newer researches have demonstrated that certain veins are supplied with constrictor fibers, such as the portal vein, which receives branches from the splanchnic nerves, and it has been suggested, and indeed almost proved, that the systemic veins have a vasomotor control. Just how much influence these vasomotor nerves have in the circulation of blood in the veins is uncertain.

IV. BLOOD-PRESSURE.

1. **Resistance of Bloodvessels.**—In a system of tubes, such as is represented by the vascular apparatus, the blood exerts equal pressure on all parts of the vessel wall at a given point, but the pressure varies proportionately with the amount of resistance the blood has to overcome.

Were the circulatory apparatus a rigid, unyielding set of tubes, the pressure would diminish in direct proportion to the distance from the heart. We have said above, that the pressure in a system of tubes depends largely on the amount of resistance which the circulating fluid has to overcome. This resistance does not arise from contact of the fluid with the vessel wall, for the fluid in direct contact with the latter is at rest, and therefore the friction must take place within the blood itself, the degree depending on the viscosity of the fluid. The amount of resistance which a constant uniform viscosity exerts is dependent on the length and diameter of the tube or vessel, and follows the law of Poiseuille. According to the latter:

$$Q = k\frac{dx}{e}h$$

Q equals the amount of fluid per second issuing from a tube, d equals the diameter, l equals the length of tube, and h equals the amount of pressure necessary to drive

blood through the vessel; k is the constant of internal friction. Hürthle has shown that this formula holds good for blood as well as for homogeneous fluids, but he claims the law is not applicable to a finely divided system of tubes like the arterial tree, on account of the elastic properties of the vessel wall. He prefers to regard the flow of blood, with decreasing blood-pressure as slower than would be deduced from Poiseuille's law.[1]

2. **Pressure in the Various Parts of the Cardiovascular System.**—There is but little difference in pressure in the larger divisions of the arterial tree, and it is not until the finest arterioles and capillaries are reached that a material decrease is observed. This decrease is still more noticeable in the veins and is lowest where the venæ cavæ empty into the heart. Some observers have claimed that pressure in the iliacs is higher than in the carotids, but Weber[2] using improved methods, demonstrates that this is impossible, and never occurs in health.

The pressure in the veins is variable, as the following table of Jakobson (Langendorff) will show:

	Mm. Hg.
V. cruralis	11.4
V. brachialis	4.1
V. facial, anterior	3.0
V. jugularis	0.2
V. anonyma	0.1

(From estimations made on sheep.)

Findlay[3] has studied in the child and in the adult, the difference between central systolic pressure and peripheral pressure, using the Riva-Rocci instrument for the former and the Gärtner tonometer for the latter. During childhood, the systolic pressure is uniform at different points of the circulation, but in adult life, the peripheral is lower than the central, this difference in pressure increasing with age. In cases of hypertension, the greater the pressure, the greater

[1] Berlin. klin. Woch., 1912, p. 773.
[2] Centralbl. f. Physiol., 1906–7. xx, p. 223.
[3] Quar. Jour. Med., 1910–11, iv, p. 489.

the difference between central and peripheral pressures.

Concerning the force which is necessary to rupture the normal artery, Gréhaut and Quinquaud[1] found that the pressure in the carotid being equal to 150 mm. of mercury, 7 to 11 atmospheres are required to rupture the vessel, or from 35 to 56 times more pressure than normally exists. As a general rule, it may be stated that arteries of smaller calibre are more resistant than arteries of larger calibre, the aorta, for instance, being ruptured by less pressure than the radial. This rule holds especially in pathological states. The pressure exerted on the human artery at the moment of rupture varies from 13 to 25 kilograms. The veins are but a little less resistant than the arteries. In pathologic states, as cerebral hemorrhage, the right carotid could be broken at 3.3 atmospheres and the left at 3.4 atmospheres. The arteries near the lesion are still less resistant, the sylvian artery breaking at 3 atmospheres.

3. **Maintenance of Blood-pressure.**—(*a*) *Energy of the Heart.* —This is a very important factor in the maintenance of blood-pressure, and plays a prominent role in its variations. All of the five inherent properties of the cardiac muscle share in this, rhythmicity, excitability, contractility, conductivity, and tonicity. Moreover, the amount of blood leaving the heart in a specified time has a relatively important share in the changes seen in blood-pressure. In general, it may be stated that increase in heart rate raises the blood-pressure, and that a lowered rate diminishes pressure. This is well illustrated by a tracing taken from Langendorff showing the effect on blood-pressure of a slow heart rate brought about by the stimulation of the vagus.

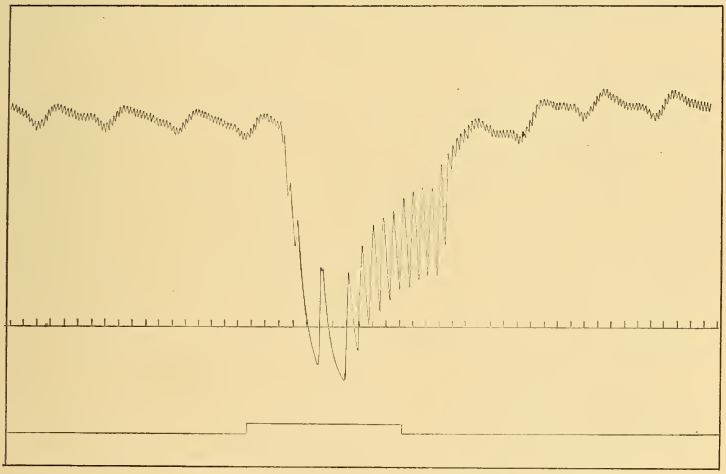
NOTE.—Experiments after cutting both vagi show that with the increased pulse rate there is an increase of blood-pressure.

There are, however, many clinical observations which do not corroborate this otherwise very simply postulate, as high blood-pressure is sometimes associated with a relatively slow heart. This is scarcely a contradiction to the general statement that increase in heart rate raises blood-pressure,

[1] Journal de l'Anat. et de Physiol., 1885, xxi, p. 287.

since we have a corollary to it in the axiom that blood-pressure depends, in addition to the heart rate, upon the amount of blood leaving the heart in a unit of time. It is readily comprehensible that a slow heart by means of its long systole can discharge a quantity of blood sufficient to counteract its pressure-lowering effects. On the other hand, it is equally true, clinically, that a rapid heart is frequently associated with low blood-pressure, as on account of the cardiac rate, there is insufficient filling of the ventricle with blood, this serving to offset the blood-raising tendencies of the rapid heart.

Fig. 2



Decrease in heart rate and fall in blood-pressure. Stimulation of vagi.

Another purely cardiac factor in blood-pressure is the strength of the cardiac systole, which, when powerful, always raises the blood-pressure. This increased force of the heart systole is generally associated with increased resistance in the peripheral circulation, or, to express it in another way, the increased force of the heart is generally a consequence of increased resistance, and not due entirely to changes in the inotropic nerve mechanism.

(*b*) *Peripheral Resistance.*—It is apparent that without resistance there can be no pressure, and it is equally apparent that the following law is applicable: the greater the resistance, the higher the pressure, other things being equal. Increased viscosity of the blood has been held to be one of the causes of increased resistance, and some observers have described a polycythemia hypertonica, the hypertension being due to the increase in the number of red blood cells. This is discussed at length in the chapter on heart and blood conditions, under the heading polycythemia. The internal friction of the blood may have a great influence in raising blood-pressure but very little evidence has been adduced as proof. The success of potassium iodide in cases of hypertension has been ascribed to its ability to diminish the viscosity, and thus lessen the high blood-pressure.

Of far greater importance is the change in the size of the bloodvessels, whereby their lumen is increased or decreased, thus diminishing or increasing the resistance respectively. The arteries are provided with a musculature absolutely under the control of the nervous mechanism which regulates the dilatation and constriction of the vessels. This vasomotor tone will be considered in more detail on page 25, but it may be said that its normal balance is most important for the preservation of good health. If the vasomotor tone is weakened through any cause, there is a great fall in pressure owing to the evident general arterial dilatation. Great depression of blood-pressure follows, due to congestion of the vessels in the splanchnic field, which, we shall learn later, is able to accommodate all the blood of the body.

Local, not extended, vascular dilatation is without effect on the systemic blood-pressure, although the reverse cannot be so positively stated.

(*c*) *Volume of the Blood.*—This plays a subordinate role, in the maintenance of blood-pressure. The volume of the circulating fluid can be much increased, without any appreciable effect on blood-pressure, provided the increase takes place slowly. This is seen experimentally by the injection

of physiological salt solution and other substances into animals. Failure, thus to raise blood-pressure, is due to three factors: (1) the rapid interchange between the vessels and tissues; (2) the automatical dilatation of the blood-vessels in order to adjust themselves to the increased amount of fluid, and (3) the subsequent polyuria. The last is the most important, the amount of urine running *pari passu* with the amount injected.

On the other hand, losses of blood do decrease, if but temporarily, the blood-pressure, so that one must, in a dog, at least, remove about one-fifth of the total quantity of blood before any great depression of blood-pressure is seen. In man, the loss of one-half of the amount of blood causes death (3–3.5 per cent. of body weight).

4. **Vasomotor Regulating Mechanism.**—(*a*) *Vasomotor Nerves.*—The smooth muscle of the arteries was discovered by Henle, and soon thereafter Stilling and Henle hinted at the presence of vasomotor nerves whose existence was later proved by Claude Bernard, in 1852. To Schiff, Ludwig, and Thiry we are indebted for the proof of the vasomotor centre.

When a nerve which controls the vessels is cut, there will be a suffusion of the area supplied by the vessels under the control of this nerve, also an increase of the warmth and size of the part and of the blood pouring from the veins. Stimulation of the same nerve has the opposite effect. Depending on the extent of the vessel's nerve distribution, there will be a fall or rise in systemic arterial blood-pressure, following severing or stimulation of the nerve respectively.

The vasomotor nerves are distributed in the musculature of the media, and since the muscle fibers are circular in their arrangement, the effect of stimulation is to constrict the lumen of the vessel (vasoconstrictor nerves). The vaso-dilator nerves are but little understood. Dilatation of the vessels has been held to be due to the influence of the longitudinal muscle fibers acting in opposition to the circular musculature, but Goltz believes that vascular dilatation is due to *inhibitory nerves*, which oppose the vasoconstrictor

fibers. It is supposed by recent investigators that both varieties of nerve fibers are present in the same nerve trunk, and that depending on the quality of the irritant constriction or dilatation is effected. This view has been held by Pal, Stricker, and others to explain the occurrence at one time of vasoconstriction and at another time of vasodilatation, as for instance, in the crises of tabes dorsalis.

(*b*) *Vasomotor Centre.*—That there is a vasomotor regulating centre has long since been definitely proved. When the spinal cord in the region of the cervical portion is severed from the medulla, the blood-pressure falls, and when the same region is stimulated there will be a rise in pressure, both phenomena being brought about by dilatation and constriction respectively, of the vessels in the splanchnic field. The seat of this vasomotor centre is in the upper part of the medulla. The centre is automatic in its activity. It is influenced principally by three factors: (1) psychic states, (2) gaseous composition of the blood, and (3) reflexly, by cutaneous influences.

(1) *Psychic Influences.*—The result of psychic influences is seen in pallor from fear, and in flushing, as a result of shame or anger, although it cannot be denied that there is an associated cardiac participation in these phenomena.

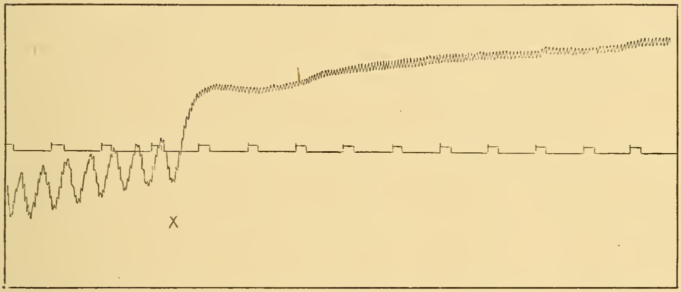
(2) *Gaseous Composition of the Blood.*—Whenever there is dyspnea the blood-pressure rises through stimulation of the vasomotor centre, some believe, from the diminished amount of oxygen and others from the increased amount of carbon dioxid. If the breath is held, the same phenomenon is observed. Fig. 3 shows the effect on blood-pressure of artificial respiration in a curarized animal (x).

When both vagi are cut, a curious phenomenon is observed, one which has been the ground for much dispute in physiological circles. Certain waves appear while the arterial blood-pressure is being registered (Fig. 4), consisting of undulations which show an ascending tendency (Traube-Hering waves). The waves are synchronous with respiration, and are intimately associated with the respiratory and vasomotor centres. Cushing[1] observed them when the vasomotor

[1] Amer. Jour. Med. Sci., 1902, cxxiv, p. 392.

centre was evidently trying to overcome whatever was producing the bulbar anemia. On comparing the waves with the intracranial tension he was able to establish the fact that the crests of the waves rose above and the hollows

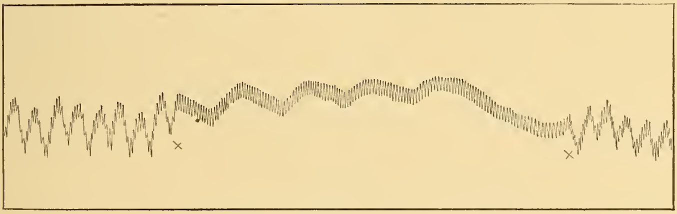
Fig. 3



Effect on blood-pressure of artificial respiration in a curarized animal.

fell below the intracranial pressure, and, furthermore, that the rise and fall of the waves was associated with blanching and reddening of the cortex. Cushing believes that the phenomenon is but an exaggeration of the normal rhythmic waves of blood-pressure seen under many conditions.

Fig. 4



Traube-Hering waves.

Wolf and Plumier[1] regard the Traube-Hering waves as an expression of rhythmical vasoconstriction extending to all the vessels of the body, arising from a stimulus sent out

[1] Journal de Physiol. et de Path. gén., 1904, p. 213.

from the respiratory centre to the vasomotor centre at each inspiration. The heart plays little part in the production of these waves.

Rhythmical variations of true cardiac origin have been described by Cushing.[1] Knoll,[2] Winterberg,[3] and Straschesko.[4]

For a full discussion on the recognition of the various waves of blood-pressure, the reader is referred to an able article by Morawitz.[5]

(3) *Reflex Influences.*—Very mild irritants applied to the skin, such as blowing on the skin, or light contact, cause a rise in pressure but strong stimuli are without effect. Certain sensory nerves, which, when stimulated, give rise to increase of blood-pressure, have a depressor function when the stimulus is applied to narcosis. Such a nerve possessing these two properties has been discovered by Ludwig and Cyon (N. depressor). It arises from the vagus and the superior laryngeal nerve, and terminates in the heart muscle itself. It is believed that vasodilatation and depression of blood-pressure follow its stimulation and Langendorff hints at the possible existence of a vasodilator centre.

(*c*) *Spinal and Peripheral Vasomotor Centres.*—The chief regulating influence of the bloodvessels lies in the vasomotor centre, whose duty it is to assume charge of all bloodvessels. The work of this centre, is, so to speak, apportioned out among lesser vasomotor centres, which regulate the activities of local vascular fields, and whose seat is in the thoracic portion of the spinal cord. This seems to be definitely proven, inasmuch as the lowered blood-pressure which follows division of the medulla, rises again, and disappears entirely when the spinal cord is destroyed. These spinal centres are excited by dyspneic and reflex stimuli but to a much less degree than the medullary centres.

[1] Journal of Phys., vol. xxv, p. 49.

[2] Sitzungsber. d. k. k. Akad. der Wissensch., Wien, 1890, Band xcix; Abt. 3, and 1894, Band ciii, Abt. 5.

[3] Zentralbl. f. Phys., 1906–7, vol. xx, p. 872.

[4] Arch. für die ges. Phys., 1909, cxxviii, p. 1.

[5] Arch. für Anat. und Physiol. Phys., Abt., 1903, p. 82.

Goltz and Ewald have shown that the arterial tone is reëstablished even after almost complete removal of the spinal cord, and it is believed that in addition to the central and spinal centres there are peripheral centres located either in the sympathetic ganglia or in the vessel walls themselves.

(*d*) *Anatomical Consideration of Vasomotor Nerves.*—According to the newer division of the nervous system into the autonomous and vegetative, whereby the former comprises the nerve distribution to the motor (muscle) portion of the anatomy, and the latter the organic portion, the vasomotor nerves fall in the class of autonomous nerves. The vasoconstrictor fibers arise in the spinal cord, and leave it through the anterior root, running in the white Rami communicantes to the gangliated cord. The cerebrospinal fiber as such, never reaches the organ, but becomes merged in the sympathetic nerve, whose termination in the organ is a sympathetic neurone. The following table gives the point of exit of nerves supplying various portions of the body:

Head: First to fifth dorsal vertebræ, especially second and third.
Upper extremity: Fourth to ninth dorsal vertebræ.
Lower extremity: Tenth to eleventh dorsal to third or fourth lumbar.
Abdominal organs: Fifth dorsal to third lumbar; kidneys especially eleventh to thirteenth dorsal.
Lungs: Second to seventh dorsal, especially third to fifth dorsal.

The vessels of the head are governed by the vasomotors in the cervical sympathetic, and destruction of the upper cervical ganglia is followed by dilatation of these vessels. Stimulation is followed by pallor, a phenomenon often observed in man. Vasomotor nerve supply to the lungs is a matter of some dispute, Wood claiming that there are pulmonary vasomotor fibres. The heart muscle receives constrictor fibers through the vagus, while the sympathetic carries dilator fibers for the coronary arteries.

All the abdominal organs receive vasoconstrictor fibers through the splanchnics, and it is for this reason that the splanchnic nerve is the most important pressure regulating nerve in the animal economy.

CHAPTER II.

VENOUS PRESSURE AND CAPILLARY PRESSURE.

I. VENOUS PRESSURE.

Although the study of arterial blood-pressure has dominated the interest of clinicians, it is perfectly comprehensible that it is, after all, but one of the many factors of blood-pressure, and that capillary, and especially venous pressure, should not be wholly neglected. The reason why venous pressure in man has not been more generally studied, is because of the lack of a method applicable to general clinical use. Simplified methods and more detailed study with accurate instruments will doubtless furnish much information of clinical value. In this chapter an attempt will be made to review the more important methods devised for determining venous pressure.

A method of estimating venous pressure by means of weights was described by Frey.[1] His idea was to compress the veins by the use of gradually increasing weights, in much the same way as one uses the fine chemical balance. The pressure read would equal the number of grams necessary to compress the vessel.

In a later paper[2] he describes a simple instrument, consisting of a piston with a spring plunger. The end of the syringe-like arrangement is placed on the vein of the arm held on level with the right third rib, and one reads off on the scale in the barrel of the syringe the amount of pressure in grams. The pressure in health is given as 10 to 15 grams.

Gärtner[3] devised a method for estimating the pressure

[1] Deutsch. Arch. für klin. Med., 1902, lxxiii, p. 511.
[2] Medizinische Wochen., 1904, p. 77.
[3] Münch. med. Woch., 1903, p. 2038.

in the right auricle. He suggested that the veins might be regarded as a manometer tube whose termination is in the right auricle, and he believed that, when the arm is slowly raised, the level at which the veins on the hands disappear, measured above the fifth rib (site of the right auricle), will give the pressure. In health the veins collapse when the hand attains the level of the fifth rib, therefore venous pressure is *nil*. In cardiac insufficiency, the arms must be raised higher to cause collapse of the veins. As ingenious as this method is, it possesses many fundamental weaknesses which do not make its usefulness an assured fact.

Oliver, and later Sewall[1] used a spring blood-pressure gauge. By this method pressure is exerted on a superficial vein, which is then milked toward the heart. The pressure is slowly released until the milked portion is seen to fill. At this instant the pressure is read off by noting the tension of a spring previously calibrated against mercury. Hooker and Eyster make objections on account of difficulty of calibration and the likelihood of mechanically raising the venous pressure distally.

The first to use air pressure was von Basch.[2] He employed for this purpose a modification of his instrument devised for estimating capillary pressure. The same principle was used by Recklinghausen[3] in an ingenious but complicated modification, with a transparent pneumatic pelotte. He found that the pressure was practically zero.

Following their leads, Hooker and Eyster[4] introduced an aluminium frame, so cut as to fit the arm, and closed below by a thin rubber sheet with a rectangular opening, and above by a glass plate. The opening in the rubber dam is placed over the vein, and by raising the pressure in the box, the vein is collapsed, the amount of pressure required to bring this about, being read in centimeters of water. With their method, the normal venous pressure averaged 8 cm. In

[1] Quoted by Hooker and Eyster.
[2] Wien. med. Presse, 1904, p. 961.
[3] Arch. für Path. und Pharmakol., 1906, lv, p. 468.
[4] Johns Hopkins Hosp. Bull., 1908, p. 274.

some cases of heart disease this was greatly raised. Accurate determination of blood-pressure is impossible when there is phlebosclerosis, a condition which Hooker and Eyster met with quite frequently.

By far the best method is the one described by Moritz and v. Tabora.[1] This is based on the principle, that if a communication is made between a vein and a manometer tube filled with water, the fluid in the latter will flow into the vein until the pressures in both become equivalent. The patient is placed in a horizontal position, the right arm is abducted to a right angle, slightly flexed at the elbow, and the hand brought in a position of half pronation. This is the position least likely to cause obstruction to the flow of blood in the veins.

A bandage is tied around the upper arm, and, observing the usual aseptic precautions, a needle is introduced into the vena mediana, and some blood withdrawn in order to see if the vein has been entered. The needle is then connected with a tubing filled with an antispetic solution (1 gram chinosol in 2000 c.c. sterile Ringer's solution) and the fluid allowed to flow into the vein until it stops, at which point the venous pressure is read off, this being the point where the pressure in the veins and the pressure in the manometer are equalized.

This method is undoubtedly very exact and occupies no more time than does an ordinary intravenous injection, and in the hands of Moritz and v. Tabora no unpleasant complications have arisen. The normal pressure corresponds to 40 to 80 mm. water, and the upper limit of normal they place at 80 to 100 mm. water. In pneumonia with decompensation the highest value was found, namely, 320 mm. water. When fully compensated, heart diseases show normal blood-pressure, but with signs of decompensation a rise is seen. In health, exertion causes a decided rise which is even more pronounced in decompensating cardiac lesions. The only valid criticism which one can make to this method is, that it is applicable only in the clinic, and its use is absolutely precluded in the consulting room.

[1] Deutsch. Arch. für klin. Med., 1909–10, xcviii, p. 475.

The latest writer on the subject is A. A. Howell[1] who seems to have devised a fairly accurate method for the estimation of venous pressure under circumstances which do not permit of the use of the Moritz-v. Tabora technique.

Howell's normal figures of 7.6 cm. of water compare favorably with those of Moritz and v. Tabora (40 to 80 mm.). In circulatory diseases with more or less decompensation, there was an elevation of venous pressure.

At present there exist very scanty references to the condition of venous pressure in disease. Judging from the prominent position the venous system occupies in sphygmographic work, it is not too much to suppose that a study of venous pressure will prove a valuable complement to arterial pressure. The nerve supply of the veins make it not at all improbable that the venous pressure undergoes considerable variation in pathological states, for it has been suggested that shock is probably a manifestation of depression of the venous pressure mechanism.

Drugs have some action on the latter, as a fall in venous pressure has been noted after nitrites and morphin.

Schott[2] has made some interesting studies with the Moritz-v. Tabora method, in observing the effect of exercise on venous pressure in various pathological states. His conclusions are, that persons with healthy hearts show no or but very little rise in venous pressure after exercise. It is possible, then, that in a study of venous pressure we may have a valuable means of estimating the functional capacity of the heart.

II. CAPILLARY PRESSURE.

The first studies in capillary pressure were by Roy and Brown on the frog, using a method whereby the pressure, necessary to make a part bloodless, is recorded. In man, v. Kries (1875) reported the first observations, using a glass plate which he pressed on the back of the finger until

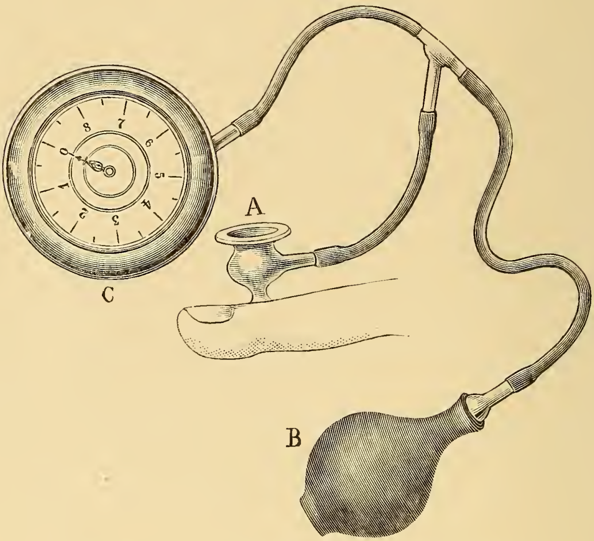
[1] Arch. Int. Med., 1912, ix, p. 149.

[2] Deutsch. Archiv für klin. Med., 1902, cviii, p. 537.

the underlying skin became pale, and then calculated the hydrostatic pressure from the weight required to blanch an area of the skin.

v. Basch[1] has made a modification of v. Kries' method, measuring the capillary pressure directly, with a manometer.

Fig. 5



v. Basche method of estimating capillary pressure.

The glass funnel, *A*, whose larger end is closed with a cemented cover glass, is placed on the dorsum of the finger, immediately behind the nail, putting a little mucilage around the skin so as to make the funnel air-tight. From *A*, a side arm leads to a *T*-tube connecting with a manometer and a rubber bulb. When the skin becomes blanched, the amount of pressure is read off. The normal pressure thus registered corresponds to 25 to 30 mm. Hg, but the arterial pressure and capillary pressure do not run *pari passu*.

[1] Wien. klin. Rundschau, 1900, xiv, p. 549.

The capillary circulation is slowest in the morning and most rapid in the evening, and this phenomenon has been held to explain the fact that the arterial blood-pressure is highest in the morning and lowest in the evening.

Lombard[1] studied the capillary pressure by a modification of v. Kries' method, and by a modification of Roy and Brown's technique. He states, that, when a gradual increasing pressure is exerted on the skin on the back of the hand, the superficial vessels are emptied in the following order: the subpapillary venous plexus, superficial venous branches, the venous arm of a part of the capillary loop (these have the lowest pressure), the arterial arm of the same loop, and the venous, and later the arterial components of the remaining capillary loops (the pressure varying in these).

He has estimated the pressures to be the following when the hand is held about 10 cm. below the second intercostal space.

	Mg. Hg.
Subpapillary venous plexus	10 to 15
Most superficial venous branches	15 to 20
Easiest compressed capillaries	18 to 22
Medium-sized capillaries	35 to 40
Most resistant capillaries and branches of the arterioles	60 to 70

These figures, he states, were obtained in normal individuals of various ages and at ordinary room temperature.

From a clinical stand-point the study of capillary pressure promises very little, at all events, the information so far obtained seems to be without much import.

[1] Zentralbl. für Phys., 1911, xxv, p. 157.

CHAPTER III.

INSTRUMENTS AND METHODS FOR ESTIMATING ARTERIAL BLOOD-PRESSURE.

I. BRIEF HISTORICAL SKETCH.

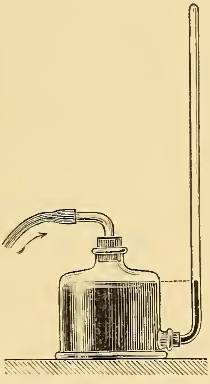
SINCE Harvey propounded his theory of the circulation of the blood, physiologists have occupied themselves with the question of estimating the degree of blood-pressure, its variations, and its relation to pathological and physiological phenomena. As early as 1733 Stephen Hales published an account of experiments made in this direction. In his Statical Essays[1] he says, "Several ingenious persons have from time to time attempted to make estimates of the force of the blood in the heart and arteries, who have as widely differed from each other as they have from the truth, for want of a sufficient number of *data* to argue from," and he adds that if they had taken their data from a regular series of experiments they would have been able to get nearer the truth. "Finding but little satisfaction in what had been attempted on this subject by Borellus and others, I endeavored about twenty-five years since to find what was the real force of the blood in the crural arteries of dogs, and about six weeks afterwards I repeated the like experiments on two horses and a fallow doe." He was the first to use the "Hydraulick Way" in making these experiments. The instrument devised by Hales was a cumbersome affair. It consisted of a brass pipe one-sixth of an inch in diameter which he inserted into the crural artery of the animal (a mare) to which was attached "by means

[1] Statical Essays; containing Hæmastaticks; or an Account of Some Hydrostatical Experiments Made on the Bloodvessels of Animals, London, 1733.

of another pipe which was fitly adapted to it" a glass tube nine feet in length and of about the same diameter as the other tube. The blood from the artery gradually rose into the tube to about eight feet, rushing up about half way at once and then ascending from twelve inches to one inch at each pulsation.

In 1829, Poiseuille replaced this long tube by a U-shaped one about 7 mm. in diameter containing mercury; the smaller arm of the U was bent at right angles and its free end was supplied with a stopcock. After being filled with bicarbonate of soda as a coagulant, it was inserted into the artery. The force of the blood-pressure was estimated by the change in the level produced in the mercury in the two tubes. Poiseuille called his instrument a hæmodynameter.

Fig. 6



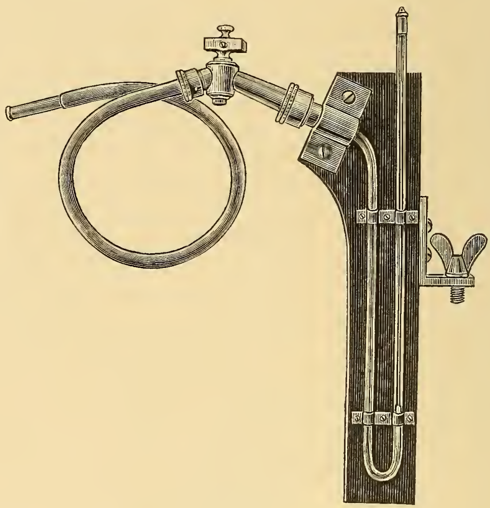
Apparatus of Guettet.

In order to facilitate the measurement, it became desirable that the movement of the mercury be limited to one tube only, and Guettet now constructed an apparatus[1] (Fig. 6) in which the mercury was contained in a flask into the top of which the tube, bent at right angles, destined to be inserted into the artery of the animal under experimenta-

[1] Morat et Doyon, Traité de Physiologie, vol. i, p. 133.

tion, was inserted, while another tube was fitted vertically to the lower side of the flask. The displacement of the mercury thus took place only in the vertical tube. The force of the pressure was estimated less the weight of the anti-coagulant used. The errors in value derived from this instrument, due to the large size of the tube, the oscillations of the mercury in the upright tube, etc., were overcome in the modification devised by Magendie (Fig. 7) which ren-

Fig. 7



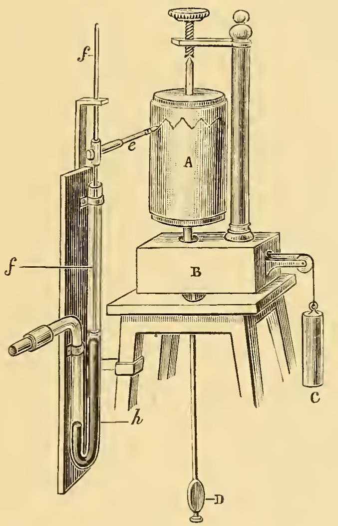
Magendie's modification.

dered the latter's model as superior to Poiseuille's as Poiseuille's was to that of Hales. In this apparatus the tubes were only about 2 mm. in diameter. While these instruments indicated, they failed to record the variations in blood-pressure, and, moreover, they required the assistance of several persons for their manipulation.

With the introduction of Ludwig's kymographion, in 1847, we have the first instrument in which the results of the blood-pressure measurements are recorded graphically (Fig.

8).[1] An ivory floater with a long light handle is introduced into a U-shaped tube. The smaller arm of the U is bent at right angles and is fitted with a rubber tube for insertion into the artery, and with a robinet for regulating the flow therefrom. The oscillations in the mercury are accompanied by oscillation of the floater, which makes its records on a paper-covered revolving cylinder. The tracings show the different waves, the smaller ones representing the systolic

FIG. 8



Ludwig's kymographion.

beats, and the larger ones being due to respiratory movements. A serious defect of this instrument was the want of continuity in the contact of the recording pencil with the cylinder. Volkmann, endeavored to remedy this by supporting the pencil with a thread. The instrument was subsequently modified by others, but the changes introduced were unimportant. The modification made by Roy, much

[1] Marey, La Circulation du Sang, 1881, p. 177.

used in laboratory work in France,[1] consists of a manometric U-tube, the smaller branch of which is supplied with rubber tubing the free end communicating with the artery. A stop-cock on the tube regulates this communication. The floater is maintained in a vertical position by a metal ring at the top of the longer arm of the U.

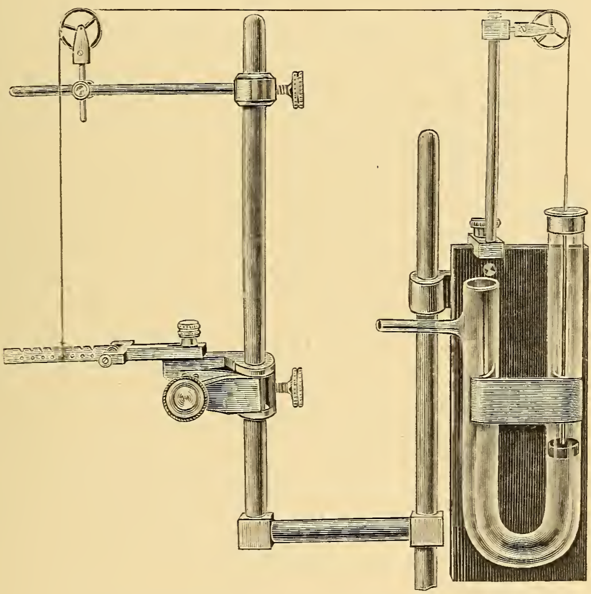
Ludwig's instrument did not give the total arterial blood-pressure, the mercury rising to the same level in both branches of the tube; total pressure was obtained by doubling the highest figure. None of these instruments so far devised registered mean blood-pressure, for it is well known that the pressure varies constantly in the blood vessels. Setschenow, and later, Marey, added value to the kymographion by an improvement by which this figure could be obtained. On the manometer of Ludwig's machine Setschenow placed a robinet at the bend of the glass tube. Closing this cock caused a gradually diminishing illumination in this part of the tube. The decreasing oscillations were thus easily seen and the level of the mercury in the larger branch remained stationary, varying only with the total blood-pressure. Making use of this principle, Marey constructed a compensating manometer which acts as a control to the graphic record of the kymographion. To a Guettet manometer flask (the one employed by Magendie and frequently known as Magendie cardiometer) he attached two tubes, one narrowed at its base to prevent too rapid movements in the mercury, and next to this he placed an oscillating column which communicates with the same reservoir. The column remained stationary between the maximum and minimum, giving the mean pressure. Another improvement was added by Chauveau, in which the floater is regulated by means of a thread passing over pulleys. At a certain point a fine pencil is affixed to the thread, and this recording point follows the movement of the thread and thus of the mercury[2] (Fig. 9).

Many of the defects of the apparatus devised since Poiseuille, were by this time remedied, but there still per-

[1] Vaschide et Lahy, loc. cit., p. 357. [2] Ibid., p. 356.

sisted the inconvenience due to the inertia of the mercury. Milne-Murray next constructed a large instrument in which the U-tube was partially filled with water. A piston, which follows the movements in the water as they are transmitted by the arterial pressure, is held by a fine wire passing over pulleys, and the recording point attached records the slightest motion in the water. The weight of the recording

Fig. 9

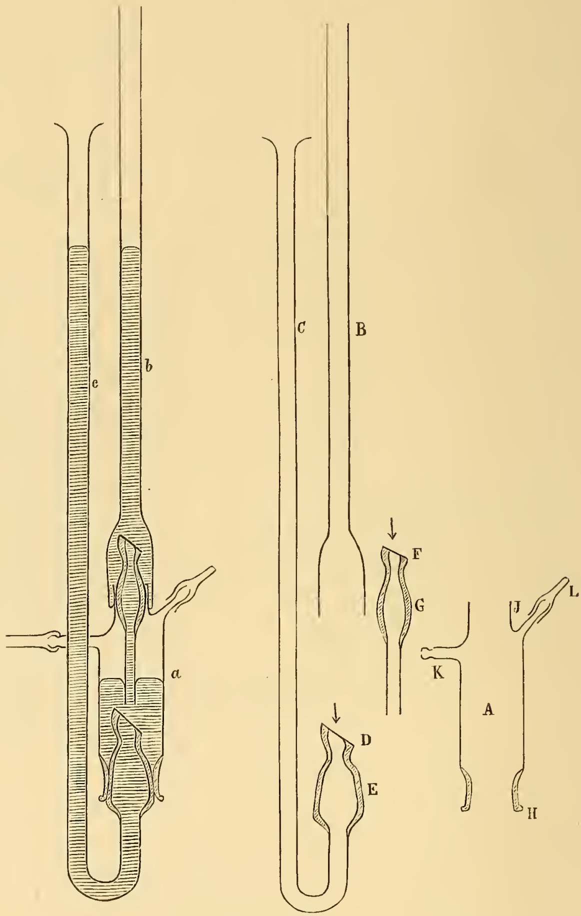


Marey-Chauveau compensating manometer.

point is sufficient to hold the thread in place. In 1878, François Franck introduced a number of important modifications into Ludwig's original apparatus and with these and some subsequent changes he may be said to have given the instrument its attainable perfection. These modifications related principally to the constant zero, which was obtained by the mobility of the graduated scale, and

to the wire thread which guides the floater, which he replaced by a hair attached at two places on one of the tubes; the

Fig. 10



Instrument of Hürthle.

management of the whole apparatus was also much facilitated by being mounted on a revolving base, and the mano-

metric tubes were also improved. Franck also devised a double manometer for recording arterial pressure simultaneously at two different points, or arterial and venous pressure at the same time.

An entirely and original instrument was at this time constructed by Hürthle—whose researches in this field are numerous and important—a maximum-minimum manometer, which, as its name indicates, registers maximum and minimum pressures. The curves of variations, however, are not recorded, and these figures must be deduced from the figures obtained[1] (Fig. 10).

As has already been stated the inertia of the mercury was a constant serious source of error in the apparatus hitherto employed, and investigators now turned their attention to the feasibility of metal manometers. Fick, in 1878, was one of the first to make the attempt, but in his endeavor to overcome the faults of the mercury he encountered a new obstacle in the instability of metallic transmission. But this source of error has been gradually eliminated and some very sensitive metal manometers, have been produced. The apparatus devised by Fick, (Federmanometer) is well known in all physiological laboratories. In this the blood-pressure acts on a tube connected with a spring (from which the instrument derives its name), which in turn communicates with a fine recording point. The instrument as first reported in a special publication[2] was afterward improved and Fick,[3] describes it as "a direct recording manometer in which, with the exception of the very light weight recording point, no inertia of any kind plays an appreciable role."

Marey's name stands forth in this connection as a methodical and original worker in this field of research. In fact, he paved the way for modern investigators, and, as will presently appear, many of the instruments widely in use at the present day are based on the principle laid down by him.

[1] Vaschide et Lahy, loc. cit., p. 359.

[2] Festschrift zur dritten Saecular Feier der med. Fac. zu Würzburg, Leipzig, 1882.

[3] Archiv für die gesamte Phys., 1883, xxx, p. 597.

In his instrument which is virtually a sphygmoscope, the blood-pressure acting on a metal capsule (of an aneroid barometer) filled with water and enclosed in a flat-bottomed vase, causes a change in the level of the alkaline solution in the upright tubes, which form part of the apparatus, and the changes are recorded by a fine point. The instrument is one in which a metallic membrane replaces a rubber one.

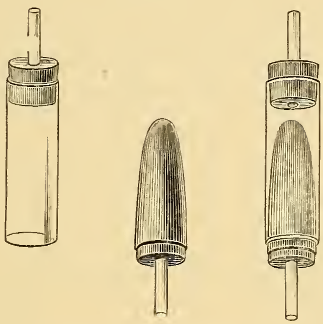
The inaccuracy of the mercurial manometer also led him to devise a simple and ingenious way of overcoming this fault, viz., the use of an elastic body for transmitting the blood waves to a metal recording manometer. This consists of a wide, short, glass tube, fitted with a cork at one end through which an ordinary piece of glass tubing extends into the wide tube; an elastic pouch, likewise pierced by a piece of glass tubing and covered with a rubber finger is introduced into the lower end of the wide glass. After being filled with an alkaline solution, and the air having been expelled from the rubber finger, its tube is inserted into the artery. The tubing at the other end of the wide glass communicates with a metal manometer. As the blood-pressure in the interior of the ampoule increases it inflates the rubber and displaces some of the air in the glass which envelops it. The variations are recorded by the fine recording point (Fig. 11).

Very important contributions to the technique of blood-pressure estimation have been made by Hürthle. Besides his numerous experiments and comparative studies with his apparatus and those of his predecessors, he has described various appliances which with more or less modifications are much used in German and English laboratories. These apparata are variously known as the spring manometer, the rubber manometer and the maximum-minimum manometer, already alluded to. Their names indicate the salient features of their mechanism.

The force of arterial pressure has been employed to unroll a torsion thread, the motion acting on a recording pencil. On this principle Roy constructed his torsion manometer. The blood enters the tube at the base of the instrument. An ebony piston held by a torsion thread closes the upper por-

tion of the tube. The torsion thread is attached to a long straight piece of steel, the length of the latter affecting the sensitiveness of the instrument. A recording point is attached to this steel rod, and when the experiment is completed the results are measured by comparison.

Fig. 11



Marey's sphygmoscope.

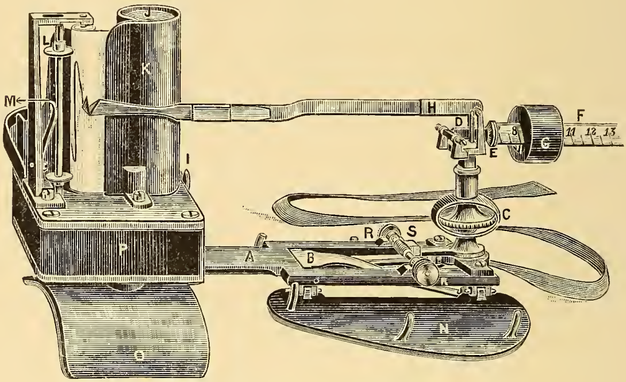
It will be readily seen that the technique for these instruments—the direct introduction of a tube into the artery—is applicable only to animal experimentation, or to man, when by chance a bloodvessel is exposed during amputation of a limb. Faire, 1856, was the first to use the method in man when he inserted a Magendie's manometer in arteries exposed during operation, but it is to Marey to whom we owe the first clinical instrument for determining arterial blood-pressure, but this was not perfected until 1878.[1]

In 1855 Vierordt conceived the idea of estimating the force of arterial blood-pressure indirectly by estimating the amount of counter-pressure required to obliterate an artery. After Marey had added the sphygmoscope to his spring apparatus, several physiologists endeavored to make use of the device for measuring the force of arterial pressure. Forster, 1867, and Behier, 1868, attempted to

[1] Shaw, Medical News, 1901, Part I, p. 373.

determine the force expressed on an artery by the spring, by means of a needle running over a dial plate set in motion by a regulating screw. At each turn of the screw the needle advanced a certain number of points. But the construction of this instrument was faulty and the results unreliable. Landois simply added weights to the spring of the syphygmograph until the artery was obliterated and he claims to have found that in students in good health it required 550 grams to occlude the radial artery, the artery being weighted for a length of 4.1 c.mm. Philadelphien's sphygmograph is another apparatus of this period in which by means of

Fig. 12



Philadelphien's sphygmograph.

weights, changeable at will, the pulse waves are registered with different known weights, the process being carried out until the pulse disappears. The weights correspond to the arterial pressure[1] (Fig. 12).

The manometers thus far described, recorded constant pressure but they failed to note variations in blood-pressure. Marey's simple and ingenious sphygmograph, it is true, indicated these modifications, but this in turn gave no information as to their extent. He himself observes[2] "the

[1] Vaschide et Lahy, loc. cit., p. 359.
[2] Circulation, p. 220, quoted by Potain, Arch. de Physiol., 1889, i, p. 559

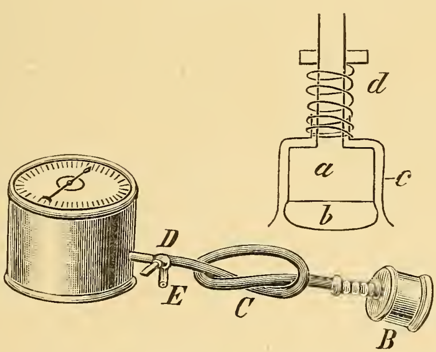
sphygmograph gives us no absolute values for the blood-pressure in the arteries, it merely expresses a relative value." Marey, and later Mosso and his followers, measured the force of arterial blood-pressure by obliterating all the blood-vessels leading to an artery.

II. MODERN INSTRUMENTS FOR CLINICAL USE.

It will be unnecessary to describe in detail, all the instruments which have been devised for measuring the arterial pressure, as a great many are now obsolete and possess only an historical interest.

v. Basch's sphygmomanometer, inasmuch as it was the first to use but a single artery for compression, may be regarded as the pioneer. This instrument was first described in 1882[1] and with it the hitherto uncertain estimation of the force of variation and amount of blood-pressure gave way to concrete figures. (Fig. 13.)

Fig. 13



v. Basch's sphygmomanometer.

v. Basch based his invention on the hydrostatic apparatus employed in animal experimentation. He eliminated springs or weighted pads as a means of compression, inas-

[1] Wien. med. Woch., 1883, p. 683; Berlin. klin. Woch., 1887, pp. 179 and 206.

much as the pressure in his instrument is exercised by means of a rubber capsule filled with water, which he terms a "fluid pelotte." This pelotte is pressed against the artery and the fluid expands according to the force of arterial pressure. The degree of expansion is recorded on a manometer connected by a rubber tube with the pelotte into which the fluid runs. In his original apparatus a mercury manometer was used, but v. Basch afterward replaced this by a metal one constructed on the principal of an aneroid barometer. Potain modified this by using air under slight pressure instead of water on the pelotte. Potain's instrument, which is much used in France, resembles v. Basch's in nearly every particular except this, but the skill required for its manipulation and the subjective relations of the operator, are defective features of the instrument.[1]

The Riva-Rocci instrument is the parent of all our modern sphygmomanometers, practically all the mercury instruments being developed from Riva-Rocci's original design. This was described at a meeting of the Italian Congress for Internal Medicine in 1896.[2] It is based on the same principle as that of the preceding instruments, namely, that the tension of a fluid in motion is in proportion to the force necessary to arrest the flow. v. Basch and Potain who made use of this phenomenon, experimented with an artery of medium caliber, while Riva-Rocci in his method used a much larger artery for compression—the brachial. The instrument consists of three parts,[3] a hollow pneumatic cuff, a pair of Richardson inflating bulbs, and a manometer. The cuff consists of a rubber tube 3 cm. or more in diameter, covered with a non-elastic material, and is supplied with a buckle arrangement for fastening it around the arm. The inner part of the cuff communicates with the manometer by means of rubber tubing and the manometer (Riva-Rocci uses a mercury manometer), and the bulbs are also connected by rubber tubing. A glass tube $2\frac{1}{2}$ to 3 cm. in diameter is placed vertically into a rather large mercury

[1] Tschlenoff, Zeitsch. für diät. und phy. Therapie, 1900, i, p. 232.
[2] La Presse Médicale, 1899, p. 37.
[3] La Tecnica sfigmomanometrica, Gazetta di Torino, 1897, No. 9, p. 184.

container 5 to 6 cm. in diameter. With these dimensions the oscillations are so slight (about 1 mm. for 300 mm. of Hg.), as to obviate the necessity for correcting errors as in the manometer of v. Basch. For use in private practice a portable metallic manometer may be used. This connects with only a single inflating bulb. The cuff is placed around the upper arm (with the buckle arrangement on the outside) and closing the cuff converts it into a genuine airless chamber around the arm. The operator palpates the pulse in the usual way. The cuff is then inflated by pressing one of the Richardson bulbs. With his eye on the manometer the manipulator gradually increases the pressure until the pulsation is entirely obliterated in the radial (or the brachial artery). In order to control the manometric indications, care must be taken to stop inflating the bulb the moment the pulse is about to disappear. The last reading is obtained by means of the second Richardson bulb. The reading may also be taken at the moment of the return of the pulse.[1] The latter usually gives somewhat lower values. Both procedures are advisable for obtaining accurate results. The figures represent maximum pressure. Minimum pressure is derived by noting by palpation the point at which the peripheral pulse waves begin to diminish. The pressure to which the veins and capillaries are subjected by this method is said to be not without danger of causing cutaneous hemorrhages in the arm and the hand, and pain, peresthesias, and other unpleasant effects may result. Moxey[2] reports a case of collapse following the use of a Riva-Rocci cuff.

The width of the cuff in the technique under consideration has been the source of much discussion. Nearly all authors agree that Riva-Rocci's original cuff is too narrow, but v. Recklinghausen goes farthest in the width of the cuff which he recommends. He finds a width of not less than 10 to 15 cm. satisfactory, but considers one of 36 cm. most useful. Few clinicians use as wide a cuff as this. The instrument is much used to this day, but nearly every operator

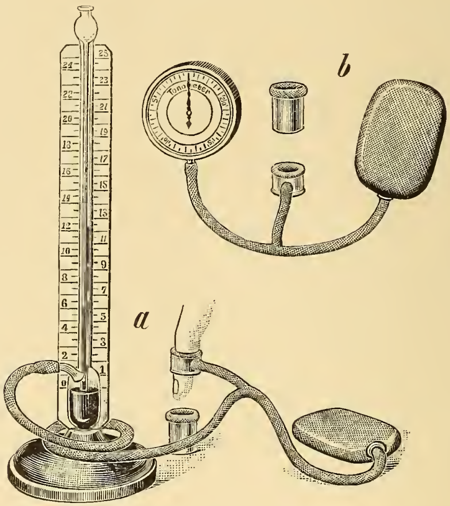
[1] Sahli, loc. cit.

[2] British Med. Jour., 1906, p. 454.

has added his own modifications to the apparatus, so that it is rarely seen in its original form.

Some of the errors in the method are due to the variations of individuals as to the amount of muscle covering the vessels under compression as well as upon the degree of thickness and rigidity of the arterial walls themselves,[1] Pachon considers the distance of the artery under compression from the region of the pulse a "characteristic and illogical feature of the method, which renders it unreliable for the determination of maximum values.

Fig. 14



v. Gärtner's tonometer.

v. Gärtner's Tonometer (Fig. 14).—This and the Riva-Rocci instrument are probably the most extensively used apparatus in modern studies on arterial blood-pressure. On account of the ease of manipulation and the simplicity of its mechanism, Gärtner's tonometer met with almost instant approval from the day of its first presentation

[1] Cushing, Boston Med. and Surg. Jour., 1903, p. 250.

before the *Wiener Gesellschaft der Aerzte*, in June, 1899, and it was at once introduced into their wards in the *Allgemeines Krankenhaus* by Nothnagel and Neusser,[1] and was favorably mentioned by many others.

The instrument consists of a pneumatic ring, a mercury manometer (v. Gärtner also constructed a portable metallic manometer for use in private practice), a rubber bulb and rubber tubing. The air-tight pneumatic ring is lined with a thin rubber membrane. The manometer is made of glass tube with a bulb in which the mercury is contained, the attached scale registers up to 260 mm. Hg. The ring, the manometer and the rubber bulb communicate with each other by means of *T*-tubing, the joints being made air-tight by means of leather washers. The ring is slipped over the second phalanx of the finger and the finger tip is blanched either by rolling a small rubber band to the joint or by placing the finger tip into a thimble-like compressor. The rubber bulb is then inflated and the figures on the manometer indicate the force with which the pneumatic ring compresses the small digital arteries. The pressure is increased until that exercised on the arteries under compression is greater than the actual blood-pressure. The rubber band is now withdrawn (or the cap removed, as the case may be), and the finger tip remains anemic; as the pressure is slowly and evenly reduced the blood rushes back as far as the ring, which prevents its further progress. The blood collecting in this area causes an intense reddening, and the patient at this moment notices a return of pulsation. The method thus differs from those already described in employing the principle that it is easier to note the return of sensation than the exact moment of its disappearance.

It has been contended that the advantage of the method lies in the fact that the sharpest sense—sight—is here employed instead of the less reliable tactile sense, but this has at the same time been set forth as a possible source of fallacies in making the readings,[2] since investigators

[1] Shaw, Medical News, 1901, Part I, p. 373.
[2] Martin, Münch. med. Woch., 1903, p. 1021.

differ as to their conception of the return of color in the fingers. Some take it at the moment of the appearance of a diffuse red, others not until the finger tip becomes deep red. In experiments on negroes, for instance, and in all cases in which artificial light was used for the experiment, it was very difficult to note the exact level at which flushing took place. The subjective sensation of the return of the blood, too, to the finger often occurs much later than the flushing; a difference of 10 to 15 mm. Hg. has been found between the reddening and the return of pulsation.

The size of the ring with regard to the girth of the finger has also been a fruitful source of controversary. Selig[1] holds that not only this but the different fingers as well as the corresponding fingers of the two hands, although of the same girth, showed variations up to 50 mm. in the manometrical record. But these variations are not as great as those due to the disproportion between the ring and the width of the finger. Nearly all criticism of the method is based on this feature as a possible source of error.[2] The law of hydrostatics, of course, makes it important that the hand be held at a level with the heart. v. Gärtner[3] offers the fact that his instrument indicates the variations due to the change of the position of the hand with regard to the heart as conclusive proof that his tonometer notes other changes as well, and that the figures obtained are an actual indication of equivalent variations in the force of the blood-pressure in the arteries. The figures correspond or come close to the absolute values of mean pressure. He proved this by experiments on dogs with white tails. Blood-pressure was estimated by the direct method and at the same time the tonometer was used on the tails. The readings of the two were taken at the same time by two different observers. The average of maximum and minimum pressure by the direct method gave the mean pressure and in each test the tonometer gave almost the same figures for mean pressure.

[1] Prag. med. Woch., 1906, xxxl, p. 87.

[2] Hirsch, Deutsch, Archiv. für klin. Med., 1901, No. lxx, p. 219. Selig, loc. cit.

[3] Münch. med. Wochenschr., 1900, p. 1197.

A practical modification of the tonometer was introduced by Federn,[1] who, using two rings connected by rubber tubing, was enabled to experiment on two fingers at the same time. v. Recklinghausen used a wider ring—3 cm. in his experiments—on the basal phalanx instead of the middle phalanx of the finger. Interesting control experiments conducted on the arm and on the fingers often showed that only a very slight difference existed between the pressure in these two regions.

v. Gärtner's tonometer has been but little used in America. The fact that articles are still published abroad, stating that the Gärtner method was the one employed, is the reason the author has devoted so much space to its description.

Stanton's instrument[2] was one of the first of its kind made in this country, and enjoyed almost universal popularity. Since Dr. Stanton's death it is no longer manufactured, and others have taken its place.

Erlanger's instrument meets all physiological requirements, but is so cumbersome that its adoption for use in office practice is out of question. A full description with illustrations may be found in the Johns Hopkins Hospital Reports, 1904, xii, p. 59.

The following instruments are enjoying considerable popularity and have the advantage of being readily portable and easy of manipulation.

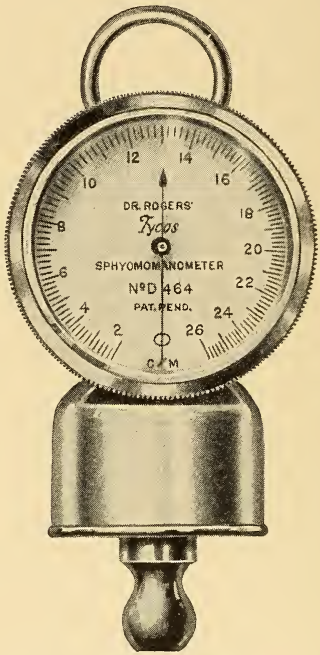
The Tycos sphygmomanometer (Fig. 15) is a spring instrument, the blood pressure being indicated by an arrow registering on a dial. It is claimed by the makers that no comparative test for the accuracy of the instrument is necessary as the hand on the dial of the sphygmomanometer rests on zero of the scale and returns to zero when the instrument is not in use. In event of injury, the hand will fail to rest on zero when the manometer is not under pressure. Its scale ranges up to 260 mm.

The special cuff which accompanies the instrument consists of a pure gum rubber band of standard width,

[1] Wolf, Wien. med. Presse, 1902, p. 1352.
[2] U. of P. Med. Bull., 1902, p. 346.

from one end of which extend two rubber tubes, the bag being enclosed in a sleeve of soft durable material. The cuff is applied, and to one of the tubes is attached the manometer, and to the other the inflating bulb. This bulb is so constructed that a control valve at the side permits the escape of air, requiring the use of but one hand

Fig. 15



The Tycos sphygmomanometer.

to raise or lower the pressure. The pneumatic system, consisting of the rubber bag with its two tubes and the diaphragm chambers of the manometer, is closed when the bulb and monometer are thus attached. Squeezing the bulb forces the air directly into the rubber bag within the sleeve, and from the sleeve as the distributing centre the air enters the diaphragm chambers, causing them to expand.

The diaphragm chambers are coupled to a wheel and a pinion by means of a connecting rod. The ratio which this wheel bears to the pinion is such, that chamber expansion

Fig. 16

The Nicholson pocket sphygmomanometer set up for use. The cuff is seen about the upper and middle third of the arm. The air pressure in the cuff and on the mercury is produced by the small pump in the operator's left hand. The right hand holds a binaural stethoscope over the brachial artery in order that the systolic and diastolic pressure may be estimated by the auscultatory method of Korotkow. The upper half of the glass tube fits into the lower half by a ground-glass joint. The mercury cannot escape because of the other valves shown by the handles.

is magnified twenty times by the hand on the dial. The elaborate details of construction are given in the circular distributed by the manufacturers.

The pressure in the cuff is raised beyond the point required to compress the artery and the air is then gradually released by means of the simple device attached to the rubber inflating bulb. The pressure may be estimated by the palpatory or auscultatory method, preferably the latter.

A mercury instrument is far more reliable than an aneroid or dial instrument, as it is more accurate and more durable. The problem in the construction of mercury sphygmomanometers has been to combine portability with the recognized accuracy and durability. Recently Dr. Percival Nicholson has devised a pocket sphygmomanometer which meets these three requisites.

New Nicholson Pocket Sphygmomanometer.—This instrument consists of a metal case which completely encloses and thoroughly protects all the metal and glass portions, making it very durable. The lid of the case when raised, automatically locks in the upright position, and acts as a support for the instrument. Full directions for the technique of using the sphygmomanometer accompany the apparatus.

Some of the advantages claimed by Nicholson for his instrument are that it has a movable millimeter scale which can be adjusted to the level of the mercury, thus removing the error incidental to changes in the mercury by barometric pressure and temperature. The use of a steel stop cock and flint glass prevents the formation of amalgam with the mercury and the employment of a steel needle valve gives perfect air release.

The entire instrument, pump and cuff, fit into a leather case which can be easily carried in the pocket. All parts are thoroughly protected so that there is little chance of damage to the parts.

III. TECHNIQUE OF ESTIMATING BLOOD-PRESSURE.
(Auscultatory Method.)

No matter which mercury instrument one selects, the essentials are the same, namely, that when the cuff is joined to the manometer and the hand pump is in position, a closed

pneumatic system is formed. When the pressure in the arm band is raised, the same amount of pressure is exerted on the mercury in the reservoir. The mercury is forced into the manometer tube, and the degree of pressure is expressed in the number of millimeters the column of mercury is raised above zero.

Three terms are used extensively in sphygmomanometry: systolic pressure, diastolic pressure, and pulse-pressure. The systolic pressure is the maximum pressure exerted on the vessel wall during a cardiac cycle. The estimation of systolic pressure should always be conducted with a wide cuff (12 to 14 cm.) as with narrow cuffs there is a great deal of variation. If a cuff 3 to 5 cm. wide is employed there will be an error of 50 mm. Hg. With a cuff 9 cm. wide there is an error of only 10 mm, but with a 12 cm. cuff the error is practically *nil.*

The pulse pressure, which is the difference between the maximum and minimum pressures, represents the intermittent burden of pressure imposed on the artery by the heart's energy in systole in order to force the blood toward the periphery and to maintain the circulation (Stone). The diastolic pressure then being the pressure in the artery due to its own constriction (systole of the artery) during diastole of the heart, pulse-pressure indicates the amount of pressure exerted by the heart in excess of the diastolic pressure. Stone believes it measures the dynamic over the potential energy and represents the load of the heart. Normally the pulse-pressure is about 50 per cent. of the diastolic pressure, the systolic being 130, the diastolic 85, and the pulse-pressure 45.

Auscultatory Method.—This method has won universal acceptance in the clinic and in the consulting room and is especially applicable to the physician's uses on account of the accurate results which it furnishes. It was described first by Korotkow in 1906.

In making blood-pressure estimations the patient may lie down or sit upright in a chair, and the patient's right or left arm may be used. It is important, indeed necessary, that successive readings should be made under the same con-

ditions, as far as circumstances permit, that is, the time of day, and the length of time after meals should be approximately the same, the patient should assume the same posture and the same arm should be used. A wide cuff having been selected, it is applied with an even pressure to the upper arm and fixed by means of straps or by folding in the small loose end according to the type of cuff employed. The tube leading from the pneumatic cuff is attached to the manometer, the arm placed in full extension and at rest and the cuff rapidly inflated by means of the hand pump or some similar appliance.

The amount of pressure exerted should always be above that of the actual blood-pressure, in other words, the pressure must exceed by 10 or 20 mm. that required to obliterate the pulse at the wrist. The degree of pressure having been attained, the air is slowly released from the cuff by means of a release screw, until the systolic pressure is learned.

For this purpose the artery at the bend of the elbow, slightly to the ulnar side, is auscultated, using a small stethoscope, and as the air in the cuff is released, a remarkable cycle of sounds will be heard. (Care must be taken to avoid all pressure with the stethoscope.) When the artery is completely compressed, nothing will be heard, but when the pressure in the cuff is decreased so that the blood is permitted to flow through the artery, the auditory phenomena begin, synchronous with each heart beat. The first sound which is heard accompanies the first column of blood which is forced through the constricted vessel. The sounds are heard with each heart beat and may be described as loud, clear-cut, snapping. This has been termed the *first phase,* after which, the pressure being slowly and gradually lowered, an entirely different series of sounds suddenly presents itself. These are murmurs, loud, rough, typical stenotic murmurs, synchronous with each heart beat, as are all the sounds with this method. This cycle is called the *second phase.* After persisting for a certain length of time, the murmurs cease as suddenly as they appeared, and give place to a cycle of sounds, which resemble those of the first phase, but are not quite so loud. This is the *third*

phase. Suddenly these sounds become less clear, and a distinct change in intensity is noted together with an altered quality, so that the new sound may be described as muted or dull. This dull quality marks the *fourth phase.* The muted sounds become still more indistinct and distant and gradually cease, and this point is called the *fifth phase.*

Originally but four phases were described, but later Ettinger detected an alteration in tonal quality of the third phase in the transition to the disappearance of all sounds and this observation having been subsequently confirmed, the normal cycle may be said to consist of five phases.

All observers agree that the beginning of the first phase—the first sound that reaches the ear—is the systolic pressure. With the auscultatory method the figures obtained are generally higher than those found with the palpatory method. There is some divergence of opinion regarding the point that marks the diastolic pressure. The fifth phase, the point at which all sounds disappear, is held by Korotkow to indicate the diastolic pressure, and with but few exceptions he has been followed in this view by all observers. Warfield has been especially active in attempting to prove that the diastolic pressure is not at the point of disappearance of all sounds (the fifth phase)—but that the true diastolic pressure is recorded when the third tone becomes dull—the fourth phase. Normally there is a difference of but a few millimeters of mercury between the fourth phase and the fifth phase, approximately 6 mm. Hg., but in certain diseases notably in cases with high blood-pressures the difference may be as great as 16 mm. Hg. Taussig and Cook also regard the fourth phase as marking the true diastolic pressure, and report some careful clinical observations to confirm their belief.

In normal individuals the determination of the diastolic pressure is a matter of the greatest ease, but in certain cases the fourth phase is totally lacking. Such cases are those with inefficiency of the cardiac mechanism. In such cases also it may be impossible to obtain accurate systolic

pressures, as a beat may be heard then no more until the mercury drops 5 or 6 mm., then a few more beats are heard, then cessation. One is unable to say at what point the true systolic pressure is indicated.

Clinical Significance of the Phases.—Inasmuch as the individual phases bear a definite ratio in health to the pulse-pressure, their significance in pathological conditions was early studied. Fischer believes the third phase is the most important, but he describes certain alterations of the phases in various diseases. In anemias, the second phase is long and plainly heard, apparently at the expense of the third phase, while the fourth phase again is longer than normal. In cases of cardiac weakness the second and third phase may both be absent, the condition being more serious in the first instance, while the presence of the third phase is associated with a less grave cardiac lesion. The third phase was found to be especially clear cut in cases of moderate arteriosclerosis and in plumbism.

These conclusions are confirmed by Ettinger, who believes that absence of the third phase is a valuable sign of cardiac weakness. In two cases there was a return of the third phase with improvement, and two cases became worse coincidentally with the disappearance of the second and third phases. In a pneumonia patient there was a sudden disappearance of the second and third phases on the day preceding death. Howell and the author[1] have also studied the length of the phases in various diseases.

Hints on Estimating Blood-pressure.—Use a wide cuff (12 or 14 cm.) and apply it to the upper arm at the level of the heart.

The cuff should fit closely but without compression.

There should be no pressure exerted by the stethoscope.

The mercury column should not be broken. With the Nicholson instrument this is readily overcome by suction. With other instruments it must be shaken down.

The instrument should not leak, and should hold the pressure without any drop in the mercury column.

[1] U. of P. Med. Bull., November, 1910; Amer. Jour. Med. Sci., September, 1911.

The patient should be at rest, and when repeated readings are made, the conditions should be as nearly the same as on previous examinations. At all events the position of the patient should be the same, either recumbent or sitting.

Two or more readings should be made, allowing time between the readings for the congestion to subside.

Individual readings should be made in as short a time as is consistent with accuracy.

When possible, blood-pressure records should be kept.

Blood-pressure readings are inaccurate when there is edema or muscular rigidity.

Sphygmobolometry.—This term was invented by Sahli to indicate a method which would measure the energy of the pulse. The method is a complicated one and is open to a great many criticisms on the basis of technic alone, if not to the criticism that it is fundamentally wrong. The author has made some investigations with the pocket instrument, and is convinced that nothing is learned by this method which is not afforded by a study of the pulse-pressure.

CHAPTER IV.

NON-PATHOLOGICAL VARIATIONS OF BLOOD-PRESSURE IN THE NORMAL MAN.

Blood-pressure in the normal young adult may be said to be systolic 120 to 130 mm. Hg. and diastolic 85 mm. Hg. (auscultatory method, wide cuff). Pressures below 120 mm. Hg. should be considered abnormal and pressures above 140 mm. Hg. are also pathologic. Females, as a rule, have a somewhat lower pressure than males, but this difference is insignificant. There is rarely a difference in pressure in the two arms. Jellinek found a variation in but 49 of 209 subjects studied. The development of the muscles of the arm and the size of the part have no influence on blood-pressure.

Influence of Age on Blood-pressure.—(*a*) **Pressure in Children.**—The younger the individual, the lower the blood-pressure, and in children the pressure varies with age, body weight and body length. General rules may be laid down as follows:

1. Blood-pressure increases with age, weight and size.
2. Blood-pressure varies in children of the same age but different height.
3. The blood-pressure is variable in children of the same age but of different body weight.
4. The sex plays but little part in pressure. It is generally from 2 to 5 mm. Hg. higher in boys than in girls.
5. Blood-pressure is increased after eating, returning to normal within two or three hours.
6. The ingestion of liquid causes a rise in arterial tension. This is an exemplification of the physiological rise of pressure after filling the stomach of an animal with water.
7. There is a rise of pressure after exercise and during nervous excitement.

Observers have found it difficult to take accurate reading in a child under three years of age, but in the main, the readings of all agree. Trumpp has made a special study of pressure in nursing babies (Gärtner instrument). The normal is about 80 mm. Hg., although the extremes of normal may be placed at 90 and 60 mm. Hg.

PRESSURE IN MM. HG. (RIVA-ROCCI).

Age.	Cook Systolic	Oppenheimer and Bauchwitz Systolic.	Wolfensohn-Kriss. Systolic.	Wolfensohn-Kriss. Diastolic.
0– 6 months	70– 75	80		
6–12 months	80– 85	90		
2– 3 years	80– 90	90	80	74
Third year	90–100			
4– 5 years	.. ..	107	83	82
6– 7 years	.. ..	..	90	82
8– 9 years	.. ..	..	90	88
6– 9 years	.. ..	111		
3–10 years	95–115			
10–11 years	.. ..	..	98	90
10–12 years	.. ..	112		
12–13 years	.. ..	..	99	95
14–15 years	.. ..	..	101	96
14 years	.. ..	158 (? ?)		
16–17 years	.. ..	..	113	105

(*b*) **Effect of Illness on Blood-pressure in Children.**—The pressure is higher in bronchitis and in pneumonia, and also in colics and in nervous excitability. There seems to be no relation, however, between blood-pressure and fever of itself. In acute gastro-enteritis there is a drop and in chronic gastro-enteritis the pressure is variable. With loss of body weight due to malnutrition there is usually a coincident lowering of blood-pressure, this occurring especially in nursing infants. In general lymphatism there is a low pressure. High pressure is the rule in nephritis, and this fact serves, according to some observers, to distinguish between this condition and lordotic albuminuria, in which the pressure is normal.

(*c*) **Blood-pressure and School Examinations.**—Putermann[1] made some observation (Gärtner) on 43 pupils, whose ages

[1] Wien. med. Woch., 1904, p. 265.

ranged between ten and sixteen years. His findings are interesting, and show that the more advanced pupils had higher pulse rate and blood-pressure before the examinations than the children of the lower classes. Furthermore, in the former the blood-pressure remained higher after the examinations, while in the latter it dropped immediately.

(*d*) **Pressure in Old Age.**—Allbutt[1] believes there is a rise of pressure in old age due to increased viscocity of the blood, and he maintains that arteriosclerosis is the result of high pressure and not the cause. He classifies the arterial diseases of elderly men into two varieties: (1) those with high pressure whose termination is death by apoplexy; (2) low-tension death by cerebral softening. Others do not regard high blood-pressure and the cardiovascular changes as a necessary effect of advancing years and hold that increased arterial pressure does not exist unless it has another cause such as nephritis and arteriosclerosis. The writer believes that the pressure rises somewhat in elderly men, but never above 150 mm., unless there is renal or arterial disease.

Influence of Temperament.—The question of temperaments is, no doubt, of importance in sphygmomanometry, but it is difficult to give any definite rule concerning it. It is generally supposed that the highly excitable individual, with high color in the cheeks and of a neurotic temperament has a higher blood-pressure than normal, but I have been surprised at the number of times this supposition has failed of corroboration. It is no doubt true that the neurotic individual responds more readily and in great degree to certain stimuli than do others of a more phlegmatic temperament.

Periodic Variations in Blood-pressure.—In the experimental animal, as well as in man, there have been observed certain constant, almost rhythmic variations in blood-pressure. Respiration plays therein an undoubted part. Lewis[2] states that in respiration there may be a fall or rise of pressure depending on whether the breathing is

[1] Lancet, 1903, i, p. 170.
[2] Jour. of Phys., 1908, xxxvii, p. 233.

intercostal or diaphragmatic, but Erlanger and Festerling[1] take exception to this and claim that there is a fall during inspiration and a rise during expiration, whether the breathing is slow or fast or abdominal or thoracic. During quiet breathing the respiratory waves are generally negligible.

Quotidian Variations.—Blood-pressure is supposed to vary during the day (many millimeters mercury), but these variations are so dependent on external stimuli that it is not fair to consider them as normal deviations. By this is meant that a stated number of individuals under the same conditions of observation, would act differently and perhaps oppositely to one another, and it would be impossible, therefore, to lay down definite rules concerning diurnal variation which would fit each individual case. Mosso[2] has made a special study of quotidian variations, which he thinks are absolutely independent of any external or psychic stimuli, and some have found pressure highest in the forenoon.

Temporary Variations.—Zabel[3] examined four healthy individuals with the greatest exactness, avoiding all conditions which would be likely to cause changes in blood-pressure, and found a great difference in systolic pressures taken at various times under exactly the same circumstances. The diastolic pressure remained unchanged. These variations have received the name psychic lability (psychogene Labilität) and certain investigators conclude that these psychic variations have a definite meaning, and indicate abnormal excitability (nervousness). A synchronous change in diastolic pressure indicates a normal circulatory apparatus, while change in diastolic pressure alone means arteriosclerosis.

The temporary changes in blood-pressure are explained by supposing increased tension of the vessel wall, and it is assumed the heart plays but little part in the variations. This opinion is based on observations following the application of electricity to the vessel wall, which is followed by

[1] Jour. Exper. Med., 1912, xv, p. 370.
[2] Arch. Ital. de Biol., 1895, xxiii, p. 192.
[3] Münch. d. med. Woch., 1910, p. 2278.

a temporary rise in pressure. Increased excitability of the vasomotor nerves and increased irritation of the intra-cardiac nervous system may also account in some way for these variations.

Rise During Measurement of Blood-pressure.—Compression of a large arterial trunk causes rise in blood-pressure, and some have raised the objection that during the act of compressing the brachial artery the blood-pressure rises and one does not obtain true values. It has been shown, however, that when the crural artery is compressed for ten seconds the response is only 2 cm. water. If the arm is compressed more than ninety seconds, the pressure rises, and he has found an increase from 140 cm. to 185 cm. water, after twenty minutes compression of the arm. Since there is some rise, one should make the blood-pressure estimation as quickly as is consistent with accuracy.

Influence of Sleep.—The changes in blood-pressure which occur during sleep have been made the subject of some research. The very elaborate technique and the care required in conducting such observations may be learned from the comprehensive article of Brush and Fayerweather. Studies were made during the whole course of sleep and it was found that the blood-pressure is lower in the late evening than in the early morning, and that during sleep pressure falls in the first few hours, rising gradually until the subject awakes. Although the increase of pressure is progressive, it is by no means regular in its ascent, the maximum height of which is reached on awakening. These observations seem to accord with those of the other observers (Kiesow, Walden, von Wagner, Colombo).

Brooks and Carroll,[1] although not using the great detail shown in Brush and Fayerweather's work, have arrived at about the same conclusions.

Influence of Posture.—When changing from the recumbent to the standing posture, the minimum pressure is increased, the systolic but little, the pulse-pressure being thus diminished.

[1] Arch. Int. Med., 1912, x, p. 97.

Erlanger and Hooker[1] have studied the velocity of blood with the v. Kries tachygraph and find that the acceleration of the blood flow per heart beat is greatest while recumbent, and least when upright. There is an inverse relation between the pulse rate, on the one hand, and the pulse-pressure and the velocity of flow, on the other. Altered hydrostatic conditions are held as the explanation of the pressure changes which accompany changes in posture. There is a rise in pressure on standing upright, but when the individual is fatigued, or if the subject is neurasthenic, a drop of 10 to 20 mm. pressure is seen.[2]

Influence of Meals.—It is difficult to form a correct estimate of the effect of taking food on the blood-pressure, for there are many factors which render a critical opinion hazardous. All the phenomena of digestion—entrance of food into the stomach, its transformation, propulsion, absorption, etc., supersede one another so rapidly that the influence of one cannot be separated from the other. Loeper[3] has attempted to avoid the inherent source of error arising from the chemical changes of the food, its molecular concentration, the amount, the irritant effect, etc. He gave a meal consisting of: 100 gm. of beef; 200 gm. mashed potato; 1 hard-boiled egg; 1 glass of water; 75 gm. of bread containing little salt.

Blood-pressure readings were made before digestion, then at half-hour intervals following the taking of food. The three phases of arterial pressure occurring during digestion are shown in Fig. 17.

In three normal subjects examined three constant curves were seen: the first consists of an elevation occurring immediately after the ingestion of food—*hypertension immédiatement;* the second, of a fall occurring fifteen to forty-five minutes after eating—*hypotension secondaire;* and the third, a rise—*hypertension tardive.* The height of the initial hypertension is seen fifteen minutes after eating,

[1] Amer. Jour. Physiol., 1903–04, x, p. 14.
[2] Richardson Medical News, 1903, lxxxiii, p. 340.
[3] Arch. des Mal. du Coeur, 1912, v, p. 225.

while the lowest point is reached about three-quarters of an hour to one and one-half hours later.

FIG. 17

Influence of food on blood-pressure.

The cause of these three phases is explained as follows: The intial hypertension is due to distention of the stomach, being especially pronounced when large quantities of liquid are ingested and persist until the liquid is expelled into the intestine.

The secondary hypotension is due to "*hypotensin*" which arises in the course of digestion. Salt and meat being the strongest secretory excitant, cause the lowest tension.

The secondary hypertension is due to intestinal distention.

This is most interesting, and inasmuch as the work has been very well done, it deserves consideration. Loeper's findings are in accord with those of Colombo, Weiss (quoted by v. Recklinghausen), and v. Recklinghausen[1] in this, that all have found the lowest pressure to occur about an hour after eating, at which time digestion is at its height.

[1] Arch. für Path. und Pharm., 1901, lxvi, p. 78.

Influence of Alcohol and Tobacco.—(*a*) Alcohol has been found to have no more influence in raising blood-pressure than the ingestion of an equal amount of any irritant. There is a change in pulse which gives the impression of increased force, but as a matter of fact it is felt at the time when there is a depression of tension, so that there is a lessening of cardiovascular activity."

It has been found that, when alcohol is introduced into the circulation, it has no stimulant action, and in moderate doses, such as are given for therapeutic purposes, it is absolutely inert as far as its effects on the heart, bloodvessel, and arterial pressure are concerned. When there is a change in pressure it is usually in the direction of a fall and not of a rise.

Another side of the question is offered by Bachem[1] in a study of the effect of wine, beer, cognac, port wine, and champagne. He found that even small quantities of alcohol speedily raise the blood-pressure, the latter reaching its maximum a half hour after ingestion. Concentrated solutions are more powerful than dilute, and the action is always more pronounced when taken on an empty stomach. This is especially true of champagne. The cause of the pressure action is not vasomotor as Kochmann believes, but is purely the result of increased cardiac activity.

It is only fair to state that in chronic alcoholics hypertension is frequently seen but this must be ascribed to arterial degeneration secondary to the toxic action of alcohol.

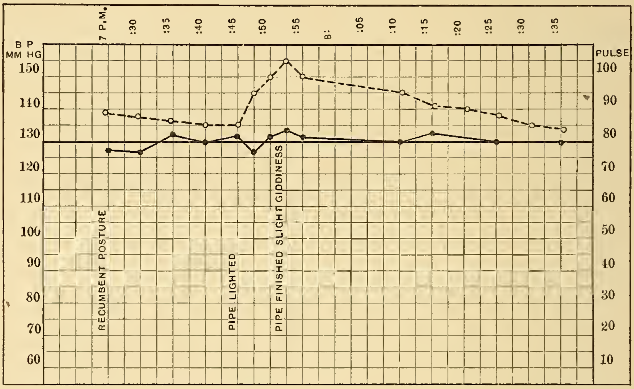
(*b*) Tobacco has a decided influence on blood-pressure, as is shown by the chart (Fig 18). Inhalation brings about a decided rise in the neophyte and in the habitué, whereas smoking without inhalation has much less effect, and sometimes none. The action of tobacco is due to the absorption of some toxic agent.

"The rise in blood-pressure is gradual, increases regularly until a rise of 10 to 30 mm. Hg. has been established and is greatest when 'strong' tobacco is used, and at the time when the smoker feels a sensation of definite intoxication. It is

[1] Arch. für die Ges. Phys., 1906, cxiv, p. 508.

most marked when the smoke of a heavy cigar is inhaled, almost as great with an old pipe, and least when the tobacco consumed is in the form of a cigarette" (Cook and Briggs). The hypertension lasts from one to two hours, after smoking, so that a smoker has constantly an abnormally high pressure. Janeway is unable to confirm the last statement.

Fig. 18



Tobacco smoking. (Cook and Briggs.)

Influence of Exercise.—When exercise is prolonged or violent, as in running, football, etc., there is a diminution of blood-pressure, but when it is moderate, the pressure is always raised. Training influences this a great deal as is shown by an interesting report by Pembrey and Todd.[1] Two men performing the same amount of exercise were studied, one of the men being in training and the other an untrained man. Both men were made to run up and down stairs for half a minute, and it was found in the trained man that the pulse-rate was doubled by the effects of exercise, but rapidly returned to its normal rate during rest, while in the untrained individual, the rate was more slowly increased and the recovery was delayed. The blood-pressure

[1] Jour. of Physiol., 1908, lxvi, p. 37.

of the athlete showed a smaller rise and a more rapid recovery than did that of the untrained man, whose pressure often fell below the normal even after a rest of five minutes.

An unique opportunity was afforded Gordon[1] to study the effects of exertion on blood-pressure in the champion club swinger of the world, who had arranged to swing his clubs for twelve hours continuously on six consecutive days. Before beginning this feat of endurance, the blood-pressure was taken, and measured 100–70. The following table will show morning and evening differences in maximum systolic pressure taken during the six successive days:

Riva-Rocci and Erlanger Instruments Were Used.

	Morning.	Evening.
First day	105	109
Second day	100	115
Third day	107	115
Fourth day	100	105
Fifth day	105	115
Sixth day	120	125

As may be seen, there was no noteworthy change in the blood-pressure. Gordon made readings also in two players in an international Rugby football match. One case fell from 145 to 120, and Gordon believes, violent exercise depresses blood-pressure, while gentle and moderate exercise tends to raise it. Otis[2] arrives at similar conclusions, as do Karrenstein, (Gärtner[3]) and Moritz.[4] The latter adds, that the more the will and attention is necessary to the exercise, the greater will be the rise in pressure. Depression of pressure below normal is a sign of cardiac tire, and in those unaccustomed to excessive blood-pressure is a good indication of how much exercise to permit.

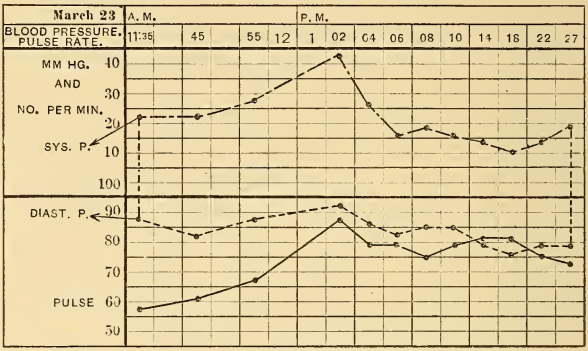
Influence of Psychic States.—Severe pain increases pressure 15 mm. Hg. (see p. 73); sensory stimuli, and a difficult mental problem raise it 20 mm.; animated conversation,

[1] Edin. Med. Jour., 1907, xxii, p. 53.
[2] Amer. Jour. Med. Sci., 1912, cxliii, p. 268.
[3] Zeitschr. für klin. Med., 1903, l, p. 322.
[4] Deutsch. Arch. für klin. Med., 1903, lxxvii, p. 339.

30 mm.; severe muscular effort 35 mm. If effort is made in absence of respiration, pressure falls.[1]

In anger, the pressure has been found by Feré,[2] to rise to great heights, and he hints at the detriment and menace attacks of cholera are to individuals suffering with disease of the arteries.[3] Feré believes anger and epileptic seizures are intimately related in their manifestations, and are physiologically not to be differentiated.

Fig. 19



Rise in blood-pressure produced by mental effort (Erlanger Sphygmogram, 12 cm.) (Janeway). Chart obtained from Prof. ———, who lectured to a large class from twelve to one o'clock. Before and after the lecture he sat quietly in his laboratory. Note the coincident rise in pulse-rate; the greater rise in diastolic pressure; the subsequent fall in pressure, with decreased pulse-pressure, while the rate remained rapid.

Janeway has constructed a chart (Fig. 19), showing the increase in systolic pressure and pulse-rate, after an hour's lecture, a rise which is obviously associated with excitement and considerable mental effort.

It is not an uncommon thing to find the blood-pressure

[1] Binet and Vaschide. Comptes Rend. Acad. de Soc. de Paris, 1897, cxxiv, p. 44.

[2] Comptes Rend. Soc. de Biol., 1889, i, p. 368.

[3] See Treatment of Hypertension.

much higher at the first visit of the patient than it is after relations are a little less new, and the marked effect which psychic states exert on blood-pressure should be watched for with great care in order to avoid incorrect interpretations of the figures obtained.

Influence of Pain.—It is difficult to estimate the influence of pain on blood-pressure, as one is often unable to exclude the influence of the conditions causing it. Some interesting observations on the changes in blood-pressure following irritation of tender points have been reported. Apart from the purely physiologic interest, these studies have some practical clinical bearing. Pressure on pain areas is followed usually by a rise in pressure, but two cases are cited by Rumuf as typical of a fall. Both were head injuries, with painful areas over the skull. In the one, the pressure was 180 mm. Hg., but after rubbing the tender spot three-quarters of a minute, the pressure sank to 156 mm. Hg. The patient had subjective pain for a considerable length of time but the pressure rose to 175 mm. Hg. a minute after the rubbing has discontinued. The other case showed a similar phenomenon:

At rest	250 mm.
Irritation of area	225 mm.
After rest	250 mm.

In any event, whether the pressure is higher or lower after irritation, the physiological normal pressure is re-attained within a very short time. Grützner and Heidenhain,[1] on the other hand, find no constant phenomena after painful stimuli.

In all these experiments, respiration and muscular contractions must be taken into consideration as tending to influence results. This seems to have been done by Curschmann, who found by using electric stimuli that there was practically always a rise of 8 to 10 mm. Hg. in normal individuals (Riva-Rocci). In patients with functional or organic circulatory disease, there was no change in pressure,

[1] Pflüger's Arch. f. Phys., Band xvi.

which was also true in cases of cutaneous paresthesias. Curschmann regards the latter observation as a proof that hysterical subjects really do have pain in the sense that those suffering with *organic* lesions have true suffering.

In tabetic crises the pressure has been seen to rise from 115 to 125 mm. Hg., to 170 to 210 mm. Hg., due to vasomotor spasm arising from the splanchnics. In lead colic the same has been observed. In pain, arising from abdominal conditions like ulcer, carcinoma, cholelithiasis, etc., the rise in pressure is very slight, not more than 10 mm. Hg.

Influence of Barometric Pressure and External Temperature.—The following report by Musgrave and Sison[1] on some blood-pressure studies undertaken in Manila, shows a lower pressure for individuals in the tropics than is found in those living in cooler climates. Although explanations are not given, the authors believe that splanchnic influences may be a prime cause, as abdominal disorders, "tropical liver" and "abdominal vacuity" would indicate. Elaborate tables may be found in their article.

The influence of temperature alone seems to have nothing to do with blood-pressure. This remains practically the same, whether there is rain or shine, whether the temperature is high or low, or whether the barometer indicate stormy or good weather. When the external temperature is lowered, however, after being high (as in bed), the blood-pressure is raised.

Effect of Compressed and Rarefied Air.—In an early work on this subject, Dietrich[2] observed that during inspiration in compressed air there was a rise of pressure in the first half of inspiration and a fall in the second half, coincidently with a smaller pulse curve as shown on the sphygmograph. The cause of the rise of pressure is to be seen in the medium pressure of the intrathoracic vessels, as in the Valsalva experiment. (Forcible expiration with nose held closed by fingers.) Expiration in compressed air, is accompanied by a fall in pressure, so that the pulse curve falls below the original base line, finally rising somewhat

[1] Philippine Journal of Science, 1910, v, p. 325.
[2] Arch. für exper. Path. and Pharm., 1884, xviii, p. 242.

as expiration is concluded. Dicrotism appears and the up stroke becomes sharper and higher, all of which indicate a rapid fall in blood-pressure. The increased rapidity of the pulse is explained on the basis of inhibition by the distended lung of the cardiac regulating mechanism.

In rarefied air, inspiration is more difficult than in normal air. Inspiration is accompanied by a slight rise in pressure, as under ordinary conditions. Expiration is accompanied by rise in pressure, which is especially noticeable toward the end of the expiration.

Müller's experiment (*inspiration* with nose and mouth closed) and Valsalva's experiment (*expiration* with nose and mouth closed) were tried. In the former, there was a sudden drop in pressure, followed immediately by a rise, which is but an exaggeration of the result obtained during inspiration in rarefied air. In the Valsalva experiment, the pressure was raised, and the pulse became slower.

Balloon ascensions have taught us that blood-pressure rises parallel to the lowered atmospheric pressure, and it is believed that this is purely a physical phenomenon and not at all a physiological one. The effects of the increased blood-pressure are not felt until there are some respiratory disturbances.[1] This view is not generally accepted, and fall of blood-pressure has been noted by experimenters using the pneumatic chamber.[2]

The difference in reports on blood-pressure during mountain ascensions is due to the difference in blood-pressure instruments used. Schneider and Hedblom[3] employed the Erlanger sphygmomanometer, while making observations at high altitudes, and they conclude that a considerable elevation in altitude tends to lower systolic and diastolic pressure and so increases the rate of heart beat. There is a greater fall of systolic than of diastolic pressure, in fact the latter may be seen to rise in some

[1] Camus: Comptes Rend. Soc. de Biol., 1903, lv, p. 790.

[2] Bert: La pressure barometrique, 1878; Lazarus and Schirmunski: Ztsch. für klin. Med., 1883; Mosso: Archives ital. de Biol., 1905, lxiii, Bartlett: Amer. Jour. Physiol., 1903–04, x, p. 149.

[3] Amer. Jour. Phys., 1908, xxiii, p. 90.

individuals, but the longer the individual remains at the high level, the more nearly normal do pulse rate and blood-pressure become. Persons who complain most of high altitudes are those in whom the greatest fall in pressure and the greatest acceleration in the rate of heart beat are seen.

Still another view is that of Staehelin[1] who believes that rarefied air has no effect on blood-pressure. This opinion is based on measurements made on himself during a balloon ascension, and while in a pneumatic cabinet.

Mountain Sickness.—Bartlett[2] transfers to man his experiments on rabbits, and concludes that there is a fall of aortic pressure when the pressure of the rarefied air falls. This is accompanied by reduction in the capacity of the arteries, and these two phenomena are followed by swelling of the lung capillaries and a lessening in the flow of blood from the lungs. Hence, in mountain sickness, pulmonary stagnation due to equalization of the atmospheric and intrathoracic pressures is the cause of the dyspnea and asphyxia, both of which symptoms are aggravated by the work which one must of necessity do in the high altitudes.

Influence of Baths.—The subject has been approached by Müller[3] in a way which seems to disarm criticism. Using the broad cuff (15 cm.) and the Riva-Rocci syphygmomanometer, he subjected three healthy men to full baths, with temperatures running from 28.7° C. (83.6 F.) to 42.5° C. (108.8° F.).

1. Baths of 33° C. (91.4° F.) to 35° C. (95° F.) and lower produced sharp increase in blood-pressure and decrease of pulse rate, both being more pronounced the colder the water became. Soon after the bath the blood-pressure became normal.

2. Baths, ranging from 33° C. (91.4° F.) to 35° C. (95° F.) to 40° C. (104° F.), cause first, an increase in blood-pressure, then a fall, and then a rise. Pulse rate is not at all constant. At 38.3° C. (100.9° F.) the fall in blood-pressure is most pronounced.

[1] Med. Klin., 1909, p. 361.

[2] Amer. Jour. of Physiol., 1903–04, x, p. 149.

[3] Deutsch. Arch. für klin. Med., 1902, lxxiv, p. 316.

3. Baths above 40° C. (104° F.) cause gradual rise in pressure with increase in pulse rate.

4. The effect of artificial Nauheim baths depends more on the temperature of the water than on carbon dioxide. A fall in pressure should be regarded seriously.

5. Sand, steam, hot air and electric light baths increase blood-pressure and pulse rate.

6. The so-called "Wellenbäder" and half baths increase blood-pressure. When the patient is very active, the pulse rate is increased also, but when the patient remains quiet it is decreased.

7. Douches of any temperature whatever increase blood-pressure.

Müller cautions that such physical measures bring about an increase of work for the heart and special care should be exercised with patients who have any organic cardiovascular diseases. Especially does the warning hold for functional cardiac disease. Many interesting figures illustrating the summary which is given above, but lack of space precludes their reproduction.

Jellinek studied some soldiers who bathed in the Danube, but obtained no uniform result. As a rule, those with low pressure showed rise, while those with elevated pressures remained unaffected.

Influence of Menstruation.—There is a fall of about 20 mm. (Riva-Rocci) when menstruation begins, reaching its lowest level at the height of menstruation. The normal level is again attained three to four days after the flow has stopped. There is an increase in pressure at puberty and also at the menopause.

Blood-pressure during Convalescence.—The blood-pressure in convalescence has received a great deal of study from Oddo.[1] The result of his research makes it apparent that during convalescence, hypotension is the rule, except in two classes of cases. The one comprises subjects showing arteriosclerosis, arthritis, nephritis, alcoholism; and the

[1] Bull. de la Soc. méd. des Hôp., May 5, 1905; Réunion biol. de Marseilles, June 20, 1905; Comptes Rend. Soc. de biol., 1905, lix, p. 719.

other includes younger subjects exhibiting what Oddo calls *erethisme cardiaque.* The latter term is applied to cases with violent cardiac impulse, accentuation of the second pulmonic, round and full, rapid, rhythmical pulse. In the majority of patients, instability of the blood-pressure is the characteristic feature, and this is associated with the common clinical observation of instabilty of cardiac rhythm. Exertion, and even change to the upright position, causes marked lowering of pressure.

The cause of the low pressure is believed to be due to asthenia cordis, and also to hypotonicity of the arterial musculature; some nerve action plays a role, also.

CHAPTER V.

PATHOLOGICAL CHANGES IN BLOOD-PRESSURE

HYPERTENSION (Hypertensive Cardiovascular Disease).

WE have learned in previous chapters of the vasomotor mechanism, by means of which blood-pressure is maintained in health at a definite level, and we have seen what changes are brought about by various stimuli, changes which, because they occur almost daily, and because they are but transient, are generally regarded as non-pathologic or functional. In normal man, blood-pressure estimated by the auscultatory method, using a standard instrument and a wide cuff, may be said to be 130 mm. Hg. (systolic) and 85 mm. Hg. (diastolic). Despite the fact that a rise in pressure in individuals past fifty is generally supposed to be physiologic, the author believes this is never above 150 mm. Hg. in health.

Permanent high blood-pressure (above 160 mm. Hg.) is met with in but two groups of cases, the one may be called simple hypertension or hyperpiesis (Allbutt) and the other is nephritis. Arteriosclerosis is purposely omitted, as we believe that the blood-pressure is but only slightly raised (140 to 150 mm. Hg.) Whenever a pressure above 150 mm. Hg. is encountered, no matter what the age of the patient, chronic nephritis should be suspected, and every means at our disposal should be enlisted before the case is classed as hyperpiesis. Schlayer,[1] in a paper on the cause of permanent hypertension, quotes some figures of Fischer, which show that of 550 patients with permanent blood-pressure above 140 mm. Hg., 62.5 per cent. had *definite* signs of nephritis, 14.5 per cent. had a *probable*

[1] Münch. med. Woch., 1913, p. 63.

nephritis, and in 23 per cent. examination was negative. Of 300 cases with blood-pressure above 160 mm. Hg., 80 per cent. showed definite signs of renal disease; in 16.3 per cent. nephritis was probably present, and in but 3.6 per cent., there was no reason for suspecting any kidney insufficiency. Of the 300 cases, 46 came to autopsy, and in all there was a definite and advanced disease of the kidneys. Such statistics substantiate the belief that pressures above normal should be regarded most seriously, not prognostically gravely, but diagnostically seriously, as nephritis is, in the vast majority of cases, the cause of hypertension, despite negative urinary findings.

Discussion of blood-pressure in arteriosclerosis will be found in Chapter VI, and nephritic hypertension will find consideration in Chapter VII, while high blood-pressure in other conditions will be spoken of in appropriate chapters. This limits us to that group of cases known as hyperpiesis, by which is meant simple hypertension without signs of renal or arterial disease. It has also received the name of hypertensive cardiovascular disease.

Krehl first called attention to this form of hypertension, and his experience has been that of all clinicians.[1] To be admitted into the class of simple hypertension (known in France and Italy under the caption "*hypertension arterielle*") there must be no clinical or anatomical evidence of a nephritis or of an arteriosclerotic process. As far as the latter is concerned, Krehl admits that conclusive evidence is almost impossible to adduce, but he states he has seen many autopsies with the aorta and mesenteric vessels showing much less change than in cases of arteriosclerosis with normal pressures. There can be no reasonable doubt that such cases of hyperpiesis do occur.

Janeway[2] has found, that in a small proportion of cases (10 to 15 per cent.) with high blood-pressure, cardiac hypertrophy and arterial changes, the hypertension is independent of nephritis. Butler[3] describes a condition,

[1] Deutsch. med. Woch., 1905, p. 1872.
[2] Amer. Jour. Med. Sci., 1906, cxxxi, p. 772.
[3] Practitioner, 1909, lxxxii, p. 854.

occurring in middle-aged and elderly people, characterized by a high blood-pressure, a tendency to moroseness or irritability and sudden apoplectiform or epileptiform attacks which leave more or less transient nervous symptoms, often aphasia or localized paralysis. He is insistent that these cases have no sign of arteriosclerosis or of Bright's disease.

As commonly seen when the heart is well compensating, the individual (generally over fifty) with hypertension is less a patient than a subject whose organism is simply under a circulatory regime and fluid equilibrium which are different from those existing in the normal state. It is a sphygmomanometric disease rather than a true disease. (Gallavardin.)

In this phase of the hypertension, even if the pressure is very high (220 to 250 mm. Hg.), there may be no symptoms; palpitation and dyspnea may be absolutely lacking. Search as one may, the individuals complain of nothing apart from perhaps slight dyspnea on going up stairs. They have nothing, and they are not patients in the strict sense of the word, that is, they are not sick. They have a large heart, and the most one can say is, that there is some hidden constitutional vice or defect which makes this large heart necessary.

So long as the left ventricle remains able to carry on its increased work, all is well. It must be remembered, however, that accidents are very prone to occur in hypertensive cardiovascular disease. Even when well compensated, cerebral hemorrhage is a common danger. The individuals are exposed to other cerebral menaces—vertigo, persistent headaches. Symptoms of renal disease may appear after the least aggravation of the hypertension—uremia.

When the heart begins to falter in its work, pulmonary symptoms are generally the first to appear. Dyspnea on slight exertion and acute pulmonary edema may disclose itself with no premonitory symptoms. Such an attack may be the first abnormal state which makes the patient consult a physician, so insidious has the weakening of the left ventricle taken place. As the cardiac arrhythmia becomes

more pronounced, dyspnea of effort is transformed into dyspnea of rest, and the patient is always short of breath.

Gallavardin writes, "but while the dyspneic episodes, caused by the faltering of the left heart, are being unfolded before our eyes, there is an organ hidden in the chest which takes on itself each blow, and which parries each with as much strength as lies in its power. This is the right heart. Good companion of the left ventricle, it is he, who gives the useful blow in return, furnishing the force necessary to cleanse the pulmonary field."

The cardiac signs of the decompensation of the left ventricle are first, gallop rhythm, then a functional murmur at the mitral area (relative mitral insufficiency), and finally, a regurgitant murmur at the tricuspid area. Tachycardia is an important sign (80 to 120) extrasystoles are sometimes heard, but the pulsus alternans is always present.

From the standpoint of blood-pressure in this stage of hypertension, there is a lowering of pressure, at first momentary and transitory, but finally becoming progressive. The diastolic pressure is generally not lowered any more than it is lowered with any fall of systolic pressure.

During the progress of cardiovascular dissolution, the kidneys have suffered in their turn and the condition now is cardiorenal and not cardiovascular alone. There is retention of chlorids and of nitrogen. Cheyne-Stokes breathing appears, insomnia, edema, and finally uremia.

The pathogenesis of this cardiovascular hypertension is not understood. A great many clinicians regard it as a latent form of chronic hypertensive nephritis, a nephritis without albumin and casts. This is not improbable, and I believe albumin and casts are sometimes a very *late* sign of a long-standing nephritis. Be this as it may, autopsy fails to reveal aught save a left-sided cardiac hypertrophy, with no sign of arteriosclerosis or of nephritis. Aubertin and Parva have made a study of the nitrogen in the blood serum of these cases but have found no increase, indicating that the kidneys are at least functionating well. Syphilis and thyroid influences may have an important bearing on the etiology.

Osler regards the high blood-pressure as salutary. This is no doubt true, but why *should* hypertension be salutary? What is the condition which so menaces the vasomotor mechanism that it must throw out this line of defense? Certain it is, that freedom from emotional states, proper diet, and rearranged course of life help in keeping the blood-pressure within moderate bounds (although the normal is rarely reached and never maintained), but this brings us no nearer the solution of the questions, what is the cause of the hypertension and wherein lies the change in these cases?

Effect of Continued Hypertension.—Despite the salutary nature of high blood-pressure it is always a serious phenomenon. The work of the heart is considerably augmented when blood-pressure has been high for any length of time, and as a result, cardiac hypertrophy is the earliest and most constant manifestation. Subjectively there may be but few symptoms, but sooner or later they appear, at first insidiously as headache, cardiac palpitation, dizziness, gastric distress, nervousness and finally, more pronounced disturbances, such as apoplexy, retinal hemorrhage, blindness, angina pectoris and acute pulmonary edema make their appearance.

On examination, the hypertrophy is readily detected, and there may be early signs of decompensation, mitral insufficiency, pulmonary stasis, ventricular extrasystoles, pulsus alternans and auricular fibrillation.

Cause of Death in Hypertension.—Heart failure in consequence of long-continued arterial pressure, must be reckoned among the causes or sudden death in hypertension. The heart failure is expressed clinically in arrhythmias, especially the pulsus alternans; all arrhythmias disappearing, however, when the blood-pressure is reduced.

Janeway[1] has studied most carefully (no autopsy records) the cause of death in 100 cases of hypertension. The clinical diagnoses were:

[1] Jour. Amer. Med. Assoc., 1912, lix, p. 2106.

Chronic nephritis	79
Diabetes	7
General arteriosclerosis	4
Coronary sclerosis	4
Aortic insufficiency	2
Aortic aneurysm	1
Primary myocardial disease	1
Paroxysmal tachycardia (with secondary myocardial insufficiency)	1
Hypertension without nephritis (evident arteriosclerosis or cardiac disease)	1
	100

The following table shows the classification of 100 cases by causes of death:

Gradual cardiac insufficiency	29
Uremic convulsions or sudden coma	15
Chronic uremia	20
Uremic psychosis	1
Cerebral apoplexy	14
Acute edema of the lungs	4
Angina pectoris	3
Sudden death (unclassified)	4
Progressive anemia	2
Acute pneumonia	4
Unrelated diseases	4
	100

The early occurrence of dysnpea in a case of hypertension indicates marked danger of cardiac inefficiency, but anginoid pains do not make the prognosis worse than do other cardiac symptoms. Complaint of polyuria, nocturnal frequency, marked headache, or of visual disturbances, especially if the patient be below fifty years of age, should make the prognosis very guarded, as uremia is frequently the mode of termination in these cases.

HYPOTENSION.

It is difficult, at the present stage of our knowledge, to state just how low the systolic pressure may fall and yet be considered normal. In an individual who has been

observed over a period of time and in whom one has determined a uniform pressure, any deviation from this standard may, with justification, bé regarded as abnormal if not pathological. But for the rank and file of individuals, it must remain a purely arbitrary judgment, at what figure to place the lower limit of normal pressure, and the standard thus erected must be regarded as but an individual opinion, nothing more.

Some draw the line between low normal and subnormal systolic pressures at 100 mm. Hg. (R. R. 5 cm.) and 90 mm. (12 cm. G.). The author prefers using the auscultatory method, to place the limit at 120 (mm. Hg.), and pressures below this should fall in the category of lowered or low blood-pressure. Such a limit is purely experimental, however, and observations made with a standard instrument, with a uniform method, and under uniform conditions, in many thousands of healthy individuals, can alone fix the limit with any degree of accuracy.

The causes of hypotension, or the conditions in which hypotension is a phenomenon may be classed as follows:

1. *Acute infections,* excepting epidemic cerebrospinal meningitis.

2. *Chronic Wasting Diseases.*—Carcinoma, tuberculosis, cachexia from whatever cause, Addison's disease.

3. *Hemorrhage, drugs, chloroform.*

4. *Nervous Diseases.*—General paralysis of the insane, neurasthenia, after epileptic seizures sometimes in tabetic crises, after lumbar puncture, Basedow's disease (at times), osteo-arthritis, exhaustion, surgical and anaphylactic shock.

5. *Cardiovascular Diseases.*—Tachycardia, dilatation of heart, arteriosclerosis (at times), mitral stenosis, decompensating cardiac lesions.

6. *Blood Dyscrasias.*—Anemia, polycythemia with splenomegaly.

7. *Renal Conditions.*—Cyclic albuminuria, nephritis (at times), amyloid disease.

8. *Intoxications.*—Alcohol, tobacco (late), acute adrenal insufficiency.

9. Terminal hypotension preceding death.

These will all be considered in appropriate chapters. There is a lamentable paucity of specific articles on low blood-pressure, the subject of hypertension apparently having dominated the interest and riveted the attention of writers. Any one who includes blood-pressure estimations in the routine examination of every patient must have been impressed with the not infrequent occurrence of blood-pressures below 120 mm. Hg., blood-pressures lying between 110 and 120 mm. Hg. Less common are the pressures lying between 100 and 110, and still more rare are those between 80 and 100, but apart from exciting surprise at the abnormally low tension, the phenomenon seems to have been little investigated. It is on account of the very brief treatment of the subject, that contributions devoted to its explanation or papers dedicated to report of cases, are extremely valuable. If one will take the pains to collect references to hypertension, he will be appalled at the amount of research performed along these lines, and will gather the incorrect and hasty impression that hypertension is after all the only problem in sphygmomanometry. It can not be denied that a persistent high blood-pressure carries in its train more destruction and more pernicious damage, and that, therefore, it deserves more consideration than does low pressure, yet this seems hardly a satisfactory explanation for the neglect which hypotension has experienced.

Its importance and significance in certain surgical conditions, notably shock, in certain clinical crises, such as anaphylactic shock, and its almost constant presence in acute fevers or infections we shall speak of in subsequent chapters. They are valuable additions to our knowledge, no doubt, but in cases other than in the above, in individuals who present no apparent organic lesion but who consult the physician about symptoms to which the specious diagnosis of neurasthenia might be too readily applied, hypotension is a subject well worth the study.

Münzer[1] endeavors to classify his cases of hypotension

[1] Wien. klin. Woch., 1910, p. 134.

in groups, and succeeds in forming six. Some of his arguments for this grouping, and the basis for his diagnosis seem to be dogmatic rather than irrefutably sound. His paper nevertheless has this value, namely, that it frankly invites criticism, its avowed object being to stimulate interest in the subject of hypotension.

The first of Münzer's groups is the hypotension seen in arteriosclerosis. He believes that this is a common cause of low pressure and offers as explanation that the blood flows through the large hard vessels, as does a fluid through an unyielding tube, there being no periodic distention of the vessel wall the tension is lowered. In some instances the pressure was as low as 85 systolic, 75 diastolic. A common trilogy of symptoms is headache, vertigo, and even fainting.

A second group of cases occurs in what Münzer believes to be status thymolymphaticus. Wiesel[1] and later Hedinger[2] observed hypoplasia of the chromaffin system in cases of this kind, and since Schur and Wiesel[3] demonstrated hypertrophy of the chromaffin system in cases of hypertension, Münzer believes one may, by analogy, expect hypoplasia to be associated with low blood-pressure. He proceeds on insufficient ground, however, to arrange certain cases in the category of status thymo lymphaticus.

The remaining groups of disease associated with hypotension are orthostatic albuminuria, chronic nephritis, paroxysmal tachycardia, and cachectic conditions.

Hypotension is by no means an uncommon feature in arteriosclerosis, as Huchard's reference in the Transactions of the Sixteenth International Medical Congress in Budapest (1919) will show. The author has such a case under his care at the University Hospital Medical Dispensary.

Thomas MacR., aged sixty-seven years; engineer. Was referred from the eye dispensary with retinal hemorrhages in the left eye. There is decided arteriosclerosis and urinary findings suggesting interstitial nephritis, namely, polyuria,

[1] Virchow's Archiv, 1904, Band 176.
[2] Ztsch. f. Pathol., 1907, i, p. 527.
[3] Deut. med. Woch., 1907, No. 51.

low specific gravity, albumin, and casts. The blood-pressure, which in this case is a variable quantity has been as low as 115 to 80 mm. Hg.

Another patient, with marked sclerotic changes in his retinal vessels, had a long standing pressure of 95 mm. Hg.

In a recent paper the author has called attention to a certain group of symptoms occurring quite frequently in cases of hypotension, and has endeavored to indicate the treatment which has been of benefit to such an individual. Full case reports exemplify these points.[1]

[1] Amer. Jour. Med. Sci., April, 1914.

CHAPTER VI.

CARDIOVASCULAR DISEASES, INCLUDING DISEASES OF THE BLOOD.

Valvular Heart Disease.—Aortic Insufficiency.—As a rule, in aortic insufficiency, the systolic pressure is high and the diastolic is low, thereby increasing the pulse pressure. There are marked variations in the systolic pressure from time to time, the cause of which is little understood, although it is held by some that such variations in other conditions are due to hypertonicity of the vessel wall. The diastolic pressure is generally lowest when compensation is failing and has been seen to rise when compensation begins to be reëstablished.

The persistence of the fourth phase (auscultatory method) is a common phenomenon in aortic insufficiency, although it is not a pathognomonic sign, it having been heard in bronchopneumonia and in Graves' Disease. The author has recently heard a persistent fourth phase in a case of arteriosclerosis. In some cases of aortic insufficiency, on the other hand, there is no persistency of the arterial sound.

The blood-pressure in the vessels of the leg and arm in health when the patient is recumbent is practically the same, but in aortic insufficiency there is a marked difference, the pressure in the leg being higher than in the arm. Leonard Hill[1] is so enthusiastic about the diagnostic importance of this phenomenon that he believes he can pick out the cases of aortic regurgitation by it alone. The following figures show striking dissimilarities:

	Arm.	Leg.
Case 1	130	195
Case 2	120	136
Case 3	150	200
Case 4	160	240
Case 5	110	130
Case 6	118	172
Case 7	130	220
Case 8	136	178

[1] Heart, 1909–10, i, 73.

Hill calls attention to the great variations which occur in the relative size of the dorsalis pedis and the posterior tibial arteries, and recommends choosing the larger for the measurement of the index.

These observations have been substantiated by Rolleston[1] who points out that well compensated cases of aortic insufficiency show a greater difference between the leg and arm pressures than do cases which have signs of failing compensations. Records are given of an interesting case of fresh endocarditis in which the leg and arm pressures were practically alike. In aortic insufficiency complicated with mitral disease, the difference is very slight.

Aortic Stenosis.—The comparative rarity of this condition probably accounts for the scant amount of blood-pressure research, although the systolic pressure is usually normal and the diastolic pressure high, giving rise to a low pulse pressure.

Mitral Insufficiency.—In a well compensated case of mitral insufficiency there is no definite change in the blood-pressure. Some writers report an increased pressure and some a decrease of tension.

Mitral Stenosis.—Korke[2] gives figures in 10 cases of pure mitral stenosis, and these range from 112 to 138 mm. Hg., irrespective of the age of the patient. The majority of cases which have come under my observation have had subnormal pressures.

The following table, copied from Gordon, will give an idea of the blood-pressure variations in the various valvular heart diseases:

Nature of disease.	Systolic.	Diastolic.	Pulse-pressure.
Aortic insufficiency	Very high	Low	Much increased
Aortic stenosis	Low	Relatively high	Much decreased
Mitral stenosis with cyanosis	High	High	Decreased
Insufficiency without cyanosis	Variable	Variable	Variable

Patent Ductus Arteriosus Botalli.—There are but two records of blood-pressure estimation, one of those is by

[1] Heart, 1912, iv, 83. [2] Lancet, 1911, ii, p. 1547

Müller[1] and the other the writer reported.[2] Müller found 150 mm. Hg. and the author found 108 mm. Hg.

Blood-pressure in Decompensation.—In the writer's experience decompensation usually brings with it a rise in arterial blood-pressure, this being due to the fact that there is increased peripheral resistance (chronic passive congestion. Decompensating heart cases with increased pressure, show a return of the latter to normal with improvement. Lang and Manswetowa have found in 17 of 18 cases of mitral disease, that during decompensation the pressure rises, and with improvement there is a fall. In cases of emphysema, these changes are especially well marked, while in aortic disease and in arteriosclerosis they are less well shown.

There is no fixed rule, however, in decompensation, as the author has had several cases with low pressure during the past year.

Non-valvular Lesions.—Acute Myocarditis.—The occurrence of myocarditis in acute infections, acute articular rheumatism, diphtheria, influenza, typhoid fever, scarlet fever, would naturally be associated with low pressure, due primarily to the action of the toxins on the vasomotor centre, and secondarily to their degenerative action on the heart muscle.

Chronic Myocarditis (Chronic Cardiac Insufficiency).—There has been an attempt on the part of certain clinicians to abandon the use of the term chronic myocarditis, claiming that this condition cannot be recognized clinically, even at autopsy it being difficult to find pathological changes.

Chronic cardiac insufficiency (dilatation) resulting from overstrain may be associated with high blood-pressure, for the retarded circulation causes stimulation of the vasomotor centre on account of the asphyxia. The resulting vasoconstriction serves the purpose of raising blood-pressure (see Chapter I) thereby relieving the asphyxia of the medullary centres, but at the same time increasing the work of the heart. It may be seen from the scheme below what a vicious circle is now operating:

[1] Correspondbl. f. Schweizer Aerzte, 1905, p. 431.

[2] Univ. Penn. Med. Bull., Dec., 1910, p. 509.

$$
\begin{tikzcd}
& \text{Cardiac weakness} \arrow[dr] & \\
\text{Increased cardiac effort} \arrow[ur] & & \text{Slower circulation} \\
\text{High blood-pressures} & & \text{through} \arrow[dl] \\
& \text{Vasoconstriction} \arrow[ul] &
\end{tikzcd}
$$

The more the arteries are constricted, the greater the work of the heart, and the more it flags, the more the asphyxia of the centres and the greater the vasoconstriction. It is comprehensible that in time the ventricle is unable to discharge all its blood at one systole and that stasis and ventricular dilatation soon result. The Germans have termed this condition "high pressure stasis" (Hochdruckstauung).

With the auscultatory method, the sequence readings may show no phases, or one phase may be lacking, usually the second or third, and with these changes, tonal arrhythimas are quite easily detected. In distinction to the functional disturbances there is no variation in successive systolic and diastolic readings which is seen in the neuroses.

Effect of Exercise in Cardiac Disease.—In degeneration of the myocardium exercise causes a rise, but this elevation is not maintained for any length of time, the pressure falling even during the exercise, though not reaching normal. During the resting period, the return to normal is lower than in health. If the rise after exercise, is but small, there is said to be a serious functional deficiency of the heart. Valvular diseases are without effect on the pressure provided the lesion be slight. When it is great the pressure behaves exactly as it does in myocardial degeneration. Moritz believes that when the pressure falls below normal after exercise it is a sign of cardiac tire (see Chapter IV, p. 71, Influence of Exercise).

Cardiac Neurosis.—The diagnosis of a cardiac neurosis is as indefinite as is the diagnosis of neurasthenia. A well-known English clinician once wrote, "Pseudo-angina is pseudodiagnosis," and the same criticism might almost be aimed at the diagnosis of "cardiac neurosis." Very little has been written about the blood-pressure in this condition,

the only work that of Hochlaus[1] but his work is little credited. Howell and the writer have studied some cases presenting the same chain of symptoms as the organic cases, but in which no organic lesion could be found, and those cases we have diagnosed as cardiac neurosis. There were marked variations in the systolic and diastolic pressures and also in the length of the phases, as apposed to a true organic case where the readings remained more or less constant. Two cases serve to illustrate this point.

Case I.—O. R., male, aged twenty years; dental student. Chief complaint: nervousness, palpitation, dyspnea, and precardial oppression, with general weakness. On examination, the heart was apparently normal. No murmurs, no arrhythmia; cardiac outlines normal. The following two readings were made within a few minutes of each other:

I		II	
145	first phase	135	first phase
120	second phase	115	
110		95	second phase
	third and fourth phase	90	third phase
P. P. 70		P. P. 45	

Note variations in successive systolic and diastolic pressures.

Case II.—Eliz. M., aged twenty-eight years. Chief complaint: cardiac palpitation, dyspnea, indigestion, headache, and nervousness. On examination, there were no signs of cardiac weakness, no murmurs, and no arrhythmia. Successive readings on the same day:

I	II
130	117
115	107
100	87
87	85
77	80

Here again are to be noted marked variations in systolic diastolic pressures, and in pulse-pressure.

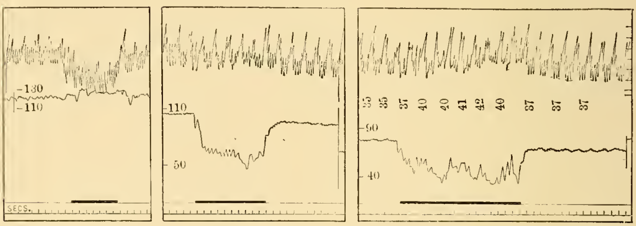
[1] Deutsch. med. Woch., 1900, p. 701.

Angina Pectoris.—During the attacks the pressure is generally high, but when the attack is over it may be as low as 80 to 90 mm. (Gärtner) (Pal.). Janeway believes one may diagnose between anginoid pain and true angina, the latter being the disease in question if the pressure is over 180 mm. (R. R. 12 cm. cuff).

Auricular Fibrillation.—Experimental studies on cats and dogs have been made by Lewis[1] in this interesting condition, by inducing fibrillation by means of faradic stimulation of the right auricle. There seems to be no constancy in blood-pressure phenomena, although there is most commonly an abrupt fall and preliminary rise, amounting to a third, or a half, of the extent of the former, and a gradually rising curve. Eventually the pressure lies within 5 or 15 mm. of the original pressure.

The venous pressure changes but slightly. As a rule, the venous and arterial pressures move divergently. Changes in both venous and arterial pressures are due to changes in ventricular contraction.

FIG. 20



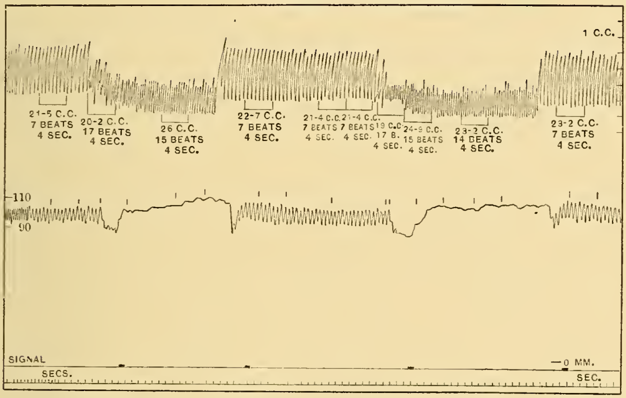
Blood-pressure in experimental auricular fibrillation.

Clinical observations on auricular fibrillation have been conducted by Silberberg[2] using an ink polygraph (Mackenzie). The inequality of the ventricular beats, producing now a strong, now a weak radial pulse, suggests some alteration in blood-pressures. And it is true, that the more irregular

[1] Jour. Exper. Med., 1912, xii, p. 395.
[2] British Med. Jour., vol. ii, p. 775.

the heart, the greater the difference in pressure. When the ventricular rate has been influenced by digitalis, the difference does not amount to more than 5 or 10 mm. Hg. The latter finding is in accord with the experiments of Lewis, to wit, that the blood-pressure changes in fibrillation are due to the changes in ventricular rate.

FIG. 21



Blood-pressure in experimental auricular fibrillation.

The records of eight cases reported by Silberberg are reproduced.

Case.	Blood-measure readings of smallest beats.	Maximum blood-pressure recorded.	Difference in mm. Hg.	Pulse rate per minute.
1	150	210	60	56
2	80	100	20	72
3	100	130	30	92
4	95	140	45	74
5	130	140	10	44 (digitalis)
6	115	140	25	84
7	120	140	20	72
8	160	200	40	76

It is obvious that the ordinary method of estimating blood-pressure is quite inaccurate, as in the extreme arrhyth-

mia seen in auricular flutter each pulse wave has a different pressure. Mackenzie (quoted by James and Hart)[1] says, "we gave up the attempt to register the blood-pressure in cases of auricular fibrillation, for though some sort of a result could be obtained, the result would be expressed by a figure, and this would have given an aspect of precision which it did not possess and would therefore inevitably mislead."

It has been suggested by James and Hart that to overcome the inherent errors of the ordinary blood-pressure estimation, one should study the average systolic pressure. To obtain this, "the apex and radial are counted for one minute, then a blood-pressure cuff is applied to the arm and the pressure raised until the radial pulse is completely obliterated; the pressure is then lowered 10 mm. and held at this point for one minute while the radial pulse is counted; the pulse is again lowered 10 mm. and a second radial count is made; this count is repeated at intervals of 10 mm. lowered pressure until the cuff pressure is insufficient to cut off any of the radial waves (between each estimation the pressure on the arm should be lowered to 0). From the figures thus obtained, the average systolic blood-pressure is calculated by multiplying the number of radial beats by the pressures under which they came through, adding together their products and dividing their sum by the number of apex beats per minute, the resulting figure is what we have called the "average systolic blood-pressure." The authors illustrate their method by the following observation:

Brachial pressure.	Radial count.
100 mm.	0
90 mm.	13 = 13 × 90 = 1170
80 mm.	47 − 13 = 34 × 80 = 2720
70 mm.	75 − 47 = 28 × 70 = 1960
60 mm.	82 − 75 = 7 × 60 = 420
50 mm.	101 − 82 = 19 × 50 = 950
	Apex = 131) 7220
	Average systolic pressure = 55 +

[1] Amer. Jour. Med. Sci., 1914, cxlvii, p. 63.

Digitalis in cases of auricular flutter is said to exert its beneficial effect by slowing and increasing the force of ventricular activity, thereby raising blood-pressure.

Paroxysmal Tachycardia.—In paroxysmal tachycardia there is a fall of blood-pressure bringing about cerebral anemia, and this accounts for the weakness, giddiness, muscæ volitantes, and fainting attacks complained of by the patient during the attack.

Functional Tests of Heart.—The estimation of the functional capacity of the heart is a matter which is far from being as simple as it at first sight appears. It is a matter of the greatest importance and, at the same time, one of the greatest difficulty, as, from a purely economic standpoint it is a vital question to an individual to know just what kind of work he is fitted for, just how much work he may do, in what form of exercise he may indulge, all without drawing too deeply on the reserve power of the heart. As Hirschfelder says, "The important question is not what the patient can do in a gymnasium, but what he can do or what he can not do in every-day life." A patient can estimate to a certain extent his cardiac limitations, for one often hears the statement, "I do not get short of breath on going up stairs, or when walking quietly, but if I hurry, I cannot breathe so easily." Therefore he does not run up stairs, not does he run for trains. In other words, functional tests, which of necessity, are directed so as to throw extra strain on the heart, express themselves in the physical signs and subjective symptoms of the patient almost as distinctly, as in the changes of blood-pressure. The functional tests with which we are best acquainted have not proven very illuminating.

Methods Suggested for Testing Cardiac Efficiency.—1. When a subject stands after having been in the recumbent posture, there is an increase in pulse rate, generally not more than twenty beats per minute. In cases with failing compensation, this acceleration is increased, so that the pulse rate rises more than twenty beats a minute. A modification of this test is noting the decrease in heart beats when the patient lies down. Normally, the slowing should be up to thirteen

beats per minute, but in the cases of cardiac inefficiency there is little, if any, difference. This test has the obvious error that too much depends on physical factors.

2. **Method of Mendelsohn-Gräupner.**[1]—On the postulate that the more functional activity a cell or an organ or an organism possesses, the more rapidly and the more completely will it replenish its store after being consumed during work, Mendelsohn bases his "Erholungsmethode." His method consists of making a patient work a wheel, which is so arranged that the brake band can be weighted, and by turning the wheel a complete revolution (one meter), the amount of work done in a unit of time can be estimated. Thus, a wheel turned once with a kilogram weight equals 1 kilogrammeter work. Mendelsohn found that with 100 to 200 kgm., the pulse rate, which is increased, falls immediately to normal (moderate work). After 200 to 500 kgm., pulse rate is normal in 2 to 3 minutes (large amount of work). Over 500 kgm., the time in which the heart recuperates depends on the extent and the degree of the exercise. In heart cases, mild exercise (25 to 50 kgm.), was followed by persistent increase of pulse rate even after exercise had been discontinued for some time. This method can be modified in various ways, as Cabot and Bruce[2] have done. The last named authors who agree with Gräupner, estimate the amount of work done in foot pounds, by having the patient walk up a measured flight of steps.

3. **Katzenstein's Test.**[3]—Katzenstein has made clinical application of the observation of Marey and Weber, namely, that ligation of a large artery is followed by rise in general blood-pressure. It was thought by the two writers that the rise in blood-pressure was due to the relative increase in the total amount of circulating blood, but Katzenstein believes it to be a result of extra work thrown on the heart by the constriction.

[1] Mendelsohn: Verhandl, des Congr. für inn. Med., 1901, xix, p. 200; Gräupner: Berlin. Klinik, 1902, xv, No. 174.

[2] Amer. Jour. Med. Sci., 1907, cxxxiv, p. 491.

[3] Deutsch. med. Woch., 1904, pp. 807 and 845.

The patient must lie quietly for a short while before the test is made, and all physical influences are to be removed. Both iliacs are compressed with the index or ring-finger at the ligamentum inguinale. When pressure is insufficient, there is a slight thrill felt, but to compress the arteries not a great deal of force is required. The compression is to be kept up for two to two and one-half minutes. Katzenstein says he has pressed for five minutes, during which time very little exertion was necessary, except in cases of arteriosclerosis. Since the blood-pressure does not reach normal for ten to twenty minutes there is plenty of time to make repeated blood-pressure readings after pressure has been released (R.R.)

	Blood-pressure.	Pulse.
Normal individuals	Rises 5–15 mm. Hg.	Stationary or falls
Hypertrophy of heart when it is efficient	Rises 15–40 mm. Hg.	Stationary
Hypertrophy of heart when it is inefficient	Does not rise 15 mm. Hg.	Rises
Mild inefficiency	Below 15 mm. Hg.	No change
Severe inefficiency	Below 15 mm. Hg.	Rises

Levy[1] confirms Katzenstein's observations, but believes the results are inaccurate when the test is applied to nervous or sensitive patients. Hoke and Mende[2] have found that not all healthy individuals react with a rise in blood-pressure and a fall in pulse rate, and also, that in severe cases of cardiac inefficiency, the method is worthless. They are emphatic in their statement that in cases of bad decompensation the method is too dangerous and should not be used.

4. **Method of Herz.**—After counting the pulse, the patient is instructed to flex his arm, the physician, meanwhile, resisting his movements to a certain extent. In health, there is but little change in the pulse rate, if any, it is slightly increased. In diseased hearts, on the other hand, there is a slowing of the beats per minute. The author gives no details as to the number of times the movements are to be made.

[1] Ztsch. für klin. Med., 1906, lx, p. 74.
[2] Berlin. klin. Woch., 1907, p. 304.

5. The author believes[1] that in the auscultatory method of estimating blood-pressure we have a fairly good index to the work capacity of the heart.

Those interested are referred to the original papers by Howell and the author quoted in the section devoted to the description of the auscultatory method. In these papers will be found the writer's views regarding the usefulness of the phases in estimating cardiac function together with cases illustrating the changes the phases undergo in decompensation.

6. **Method of Schott.**—Schott[2] makes use of the venous pressure, using the method of Moritz and v. Tabora, which has been described in the chapter on venous pressure. Schott constructed an apparatus so that bed-ridden patients could perform a certain amount of work, after the completion of which blood-pressure is estimated. His observations are grouped as follows:

1. Healthy individuals exhibit very little or no elevation in the venous pressure (0.5 cm. water).

2. Patients with valvular lesions, or myocarditis, with moderate subjective symptoms, but who could not be said to have decompensation, showed an average rise of 2.3 cm.

3. Patients who were admitted with decompensation but who, under treatment, became so much better that the edema entirely disappeared, had a rise of 47 cm. water.

4. Cases with decompensation, namely, edema, chronic passive congestion of liver, albumin, the venous blood-pressure rose 7.3 cm.

It will be seen that the more severe the decompensation the higher will be the rise of blood-pressure.

This method of Schott, while apparently very accurate, has the great drawback, that it is of use only in the well-equipped clinic, and that its employment elsewhere is absolutely precluded.

The objection to all these methods used for testing the cardiac function is that they are not more delicate than the

[1] Amer. Jour. Med. Sci., September, 1911.
[2] Deutsch. Arch. für klin. Med., 1912, cviii, p. 737.

simpler clinical manifestations, such as respiratory distress, cyanosis, decrease in size of pulse, tachycardia, and arrhythmia.

Russell[1] believes that the extreme variations of blood-pressure seen in the same individual are due to hypertonic contraction of the bloodvessel walls, and not to increased work of the heart. He claims, therefore, that blood-pressure estimates of functional capacity of the heart are fundamentally wrong, although Janeway and Park[2] state that a "hypertonic contraction of more than 30 mm. Hg. seems improbable and of more than 60 mm. incredible during life."

Arteriosclerosis.—A priori, one would say that arteriosclerosis is a common cause of hypertension, but on closer reflection, it must be debated whether arteriosclerosis is a cause of high blood-pressure, or if high blood-pressure is a cause of the arterial degeneration. Huchard, in his work on diseases of the heart and aorta, thinks that arteriosclerosis *is* a cause, while Thoma[3] holds that in an uncomplicated degeneration of the arteries, blood-pressure does not rise. It remains true, nevertheless, that in cases of arteriosclerosis with hypertension, even though clinical signs of a renal complication fail, one is not justified in claiming that no nephritis is present. On the other hand cases of hypertension do occur without any kidney lesion, notably in Graves' disease, melancholia, neurasthenic states, and diseases of the central nervous system. This view is substantiated by Krehl[4] who found at autopsy many cases of high blood-pressure with no arterial degeneration (see Chapter V). However, nephritis must be considered as the principal cause of hypertension, although there are often seen cases of arteriosclerosis, who have had during their lifetime, high blood-pressure, but who exhibit no pathologic changes of the kidney at autopsy.

Sawada[5] examined 206 cases of arteriosclerosis (R.R.),

[1] British Med. Jour., 1912, i, p. 659.
[2] Arch. Int. Med., 1910, vi, p. 586.
[3] Virchow's Archiv, 1886, civ, pp. 209 and 401.
[4] Deutsch. med. Wochensch., 1905, p. 47.
[5] Ibid., 1904, No. 12.

fixing the limit of blood-pressure in health at 120 mm. Of 98 cases of arteriosclerosis having normal heart and kidneys, there were 83 with normal pressure, 10 border-line cases (120 to 130 mm. Hg.), and 5 with pressures above 130 to 140 mm. Hg. Only one patient had a blood-pressure of 176 mm. Hg., and Sawada was not able to exclude nephritis in this instance. He divides, purely arbitrarily, arteriosclerosis into three grades, the first with palpable thickening of the wall, the second when the artery is distinctly palpable, and the third grade when the vessel has a decided pipe-stem feel.

First group 42 cases . .	3 (120–130) 2 above 130 mm. Hg.
Second group 42 cases . .	5 (120–130) 2 above 130 mm. Hg.
Third group 14 cases . .	2 (120–130) 1 above 130 mm. Hg.

He concludes, since only 12.3 per cent. of arteriosclerotic individuals show a slight increase of pressure (130 to 160 mm. Hg.) that arteriosclerosis alone can not account for the hypertension. He believes that values above 160 to 170 mm. Hg. point definitely to an interstitial nephritis, even when there is no albuminuria and no cylindruria.

The conclusions of Sawada, are those of every one who has made routine blood-pressure examinations, and every clinician has seen cases of marked arteriosclerosis with no cardiac hypertrophy and no evidence of hypertension. Brault takes a decided stand in the matter and disclaims any influence on blood-pressure of an arteriosclerotic process, insisting that any cardiac hypertrophy which ensues, is due to a concurrent nephritis.

In a careful anatomical study, Hasenfeld[1] has shown that arteriosclerosis of the splanchnic vessels is not at all uncommon, but that there is rarely the degree of degeneration which one finds in the aorta, in the vessels of the extremities, and in the brain. Arteriosclerosis, even when general, brings with it no cardiac hypertrophy unless the splanchnic arteries are involved or the aorta above the diaphragm is markedly diseased. Hirsch[2] has confirmed fully Hasenfeld's

[1] Deutsch. Arch. für klin. Med., 1897, lix, p. 193.
[2] Ibid., 1900, lxviii, p. 55.

observations and has offered further proof of the correctness of the latter's views by reporting 5 cases of marked hypertrophy and pronounced splanchnic arteriosclerosis.

These views of Hasenfeld and Hirsch have been taken exception to, the claim being that no constant relation exists between left-sided hypertrophy and changes in the celiac and mesenteric arteries. In the vast majority of cases, an associated contracted kidney can be held as the cause of the cardiac hypertrophy. Arteriosclerosis of the thoracic aorta can not be a constant cause, either such changes in the aorta with no hypertrophy of the heart, and hypertrophy of the heart with no changes in the thoracic aorta do occur.

Longcope and McClintock[1] constricted the superior mesenteric artery or the artery and the celiac arteries, and studied the effect on blood-pressure and on cardiac changes. The dogs were studied for a period of five months and during this time there were no changes in blood-pressure and no cardiac hypertrophy. They also say that there can be found in man no definite association between sclerosis of the abdominal aorta and great splanchnic vessels and cardiac hypertrophy.

If Hirsch and Hasenfeld's views were correct, it would have received full confirmation by this piece of experimental work, and hence we must regard sclerosis of the mesenteric vessels as of little significance in the production of cardiac hypertrophy.

An important sign of sclerosis is said to be the "Stauungsreaktion." Hertzel[2] compresses the artery in both legs and in the arm, and in the other arm he measures the blood-pressure. In health there is supposed to be a rise of 5 mm. Hg., while in arteriosclerosis, the rise is 60 mm. or more. The explanation offered is, that in the diseased arteries, owing to the congestion induced, there is a compensatory dilatation with resulting rise in blood-pressure.

Thoracic Aneurysm.—It is a well-established statement which has found its way into every text-book, namely, that there is inequality of the pulses not only as to time,

[1] Arch. Int. Med., 1910, vi, p. 439.
[2] Berlin. klin. Woch., 1913, p. 535.

but also as to volume of the artery, in aneurysm. This is chimeric in a great many instances, that is, as far as the digital perception of changes is concerned, and hence it is of most importance that blood-pressure estimations should be made to determine whether the differences so often noted are imaginary or real.

The arterial pressure in most cases of aneurysm of the thoracic aorta or innominate is either normal or slightly raised. As a rule, it is much higher in cases of simple dilatation of the aorta, a fact which promises some assistance in the differential diagnosis of the two conditions. In the majority of cases, there is a difference of at least 5 mm. between the pressure in the two arms in cases of aneurysm and in simple dilatation, while a more marked difference is equally frequent in both. A difference of 20 mm. is common in the two classes of cases and hence there is little diagnostic value in comparative blood-pressure estimations. When a difference of 30 mm. is observed, it speaks strongly in favor of aneurysm as against mere dilatation of the aorta.

A difference of 5 or 10 mm. is found more commonly in aneurysm than in arteriosclerosis or mediastinal tumor, but is found in the last two named conditions so frequently that the blood-pressure is of little help in diagnosis. When differences of 20 mm. are obtained repeatedly evidence is in favor of aneurysm.

In cases where none of the above named pathological conditions is present (arteriosclerosis, aneurysm, mediastinal tumor), there is no distinct difference between the pressure on the two sides.

In cases with unequal pressure in the two arms, there is a large pupil on the side of the lower pressure and *vice versa.* The unequal pupils are said to be due to inequality in the pressure of the opthalmic arteries.

Anemia.—In anemia, even with great weakness, the blood-pressure may remain normal. In anemia due to hemorrhage, and in the anemia of cachectic states, the blood-pressure is low, but it is more proper to speak of the blood-pressure in these cases as a result of the *cause* of anemia and not a result of the anemia itself.

Polycythemia.—Geisböck[1] presented some cases, in 1904, of increase in the number of red-blood corpuscles, associated with arterial hypertension, which he called polycythemia hypertonica. Naegeli in his book on blood diseases has accepted this statement as true, and easily explains it on the increased viscosity of the blood. Others believe, on the contrary, that when hypertension occurs it is an indication of arteriosclerosis or nephritis, rather than a phenomon of the polycythemia. Möller[2] has collected 59 cases of polycythemia with splenic tumor, in 21 of which blood-pressure readings (Riva-Rocci) had been reported. The pressure in these cases may be classified as follows:

9 cases below 130 mm. Hg.
4 cases between 130 and 150 mm. Hg.
7 cases between 150 and 180 mm. Hg.
1 case above 180 mm. Hg.

Winter published 19 cases of polycythemia, of which, but 1 had splenic tumor. His findings are tabulated:

6 cases between 130 and 150 mm. Hg.
8 cases between 150 and 180 mm. Hg.
1 case above 180 mm. Hg.
4 cases no pressure readings given.

In the 18 cases of Geisböck, 10 were between 180 and 250 mm., 7 between 150 and 180, and only 1 was below 150 mm.

A critical study of all these cases shows no relation between the blood-pressure and the erythrocyte count, for some patients with very high blood-pressures had fewer red blood cells than with small number of corpuscles.

Möller made a study of all cases of hypertensions coming to the polyclinic in Berlin, and found of the 35 patients that in 20 instances the erythrocytes were found to be between five and six millions. He calls attention to some cases of "polycythemia" quoted by the above authors, in which

[1] Deutsch. Arch. für klin. Med., Band lxxxiii; Verhandl. des Kong. für inn. Med., 1904.

[2] Deutsch. med. Woch., 1908, p. 1888.

there was no higher count than this and recommends that the term "polycythemia" be employed when the blood count is six millions or more. In only 2 of the 30 cases, was this count observed, so there can be no direct relation between the number of erythrocytes and the blood-pressure. There is very little in the literature concerning what in America we understand under the term polycythemia (or as it is called variously Osler's disease, Vaquez's disease, erythemia, polycythemia with cyanosis, megalosplenic polycythemia), a condition characterized by increase in the number of red cells, chronic cyanosis, and enlarged spleen. The author reported one case,[1] a typical case of polycythemia, studied with the auscultatory method. Up to the appearance of this paper, no case had been studied by this method, and since in anemia the phases are loud and clear and well-marked, by analogy it might be anticipated that in polycythemia the opposite should obtain. The patient William B., came to the dispensary complaining of pain in the head and buzzing in the ears. On examination, he was found to have chronic cyanosis, splenomegaly and chronic cyanosis, and his blood count showed hemoglobin 100 to 110 per cent., red-blood cells 8,000,000 to 11,000,00. The blood-pressure was, on two occasions 128 to 105, and 114 and 93 respectively, and as far as the phases were concerned, our surmise was correct, as there was no differentiation possible. Instead, the sounds had a sticky quality which defies description. At the time we reported this case it was the first one on record which had been studied with the auscultatory method.

Watson-Wemyss[2] reports a pressure of 108 mm. Hg. in a case of Vaquez's disease.

An increased number of red-blood cells of itself does not cause hypertension.

[1] Goodman and Howell, Amer. Jour. Med. Sci., September, 1911.
[2] British Med. Jour. 1913, i, p. 702.

CHAPTER VII.

BLOOD-PRESSURE IN RENAL CONDITIONS.

It were the greatest folly on the part of the internist to erect an anatomical classification of renal conditions based on clinical experience alone. Pathologists have long disputed the anatomical entity of chronic parenchymatous and chronic interstitial nephritis, and if they fail to agree on the lesion, having every facility of examination at their disposal, it seems presumptious for the clinician to diagnose one or the other type. Widal has lately advocated a classification based on *functional* disturbance of the kidneys, a step forward in the proper direction. He has studied the excretion of urinary constituents, and bases his classification on the retention of sodium chloride or urea, respectively. The first is called chloruremic syndrome, and, clinically, is recognized by retention of chlorides and by edema. The second is called azotemic syndrome, and consists essentially of retention of urea, high blood-pressure, polyuria, and little or no chloride retention and little or no edema.[1]

Widal's types may persist to the end as individual types, the uremia which develops, being termed chloruremia and azotouremia, respectively. Sometimes the two exist together, and one sees a blending of the clinical manifestations of both. The type, as will be seen, corresponds in the main, to the parenchymatous and interstitial varieties, but Widal's classification has the merit of naming clinical conditions in terms of function and not of structure. One is forced, however, at present, to conform to the established medical nomenclature, and it will be many years before old terms cans be abandoned for new ones.

[1] La Presse Méd., November 20, 1912.

Acute Nephritis.—There is a great divergence of opinion regarding the blood-pressure in acute nephritis, although one is surprised at the dearth of articles devoted to its study. Many clinicians believe that the pressure is normal or but slightly elevated, but majority hold that hypertension is the rule. In the early days of an acute attack, the cardiovascular system may have undergone no change, but usually at the end of the second or third week the blood-pressure is raised, and by the end of the fourth week there is a definite hypertrophy of the left ventricle, with accentuation of the second aortic sound.

In nephritis, occurring in the course of an acute infection, the blood-pressure is apt to be lower than when the nephritis arises from no apparent cause. The diagnosis of acute nephritis, in the absence of an ascertainable cause, must be made with some caution, and one should never cease to entertain the belief that it is but an acute exacerbation of a latent or quiescent disease. A chronic nephritis is of such easy stages that it becomes well established before it makes its presence known, and it can well be that some of the acute attacks are but an acute inflammation of a chronic process. This is illustrated in the case of a young woman, aged twenty years, who was referred to me by her physician in order to discover the cause of a troublesome form of stomatitis, associated with a very offensive odor. The blood-pressure was 168–100 mm. Hg., there was a faint trace of albumin with but few casts and a low specific gravity urine. The heart was enlarged to the left with a decided accentuation of the second aortic. Since a year ago, the pressure has risen steadily until it is now 180–100 mm. Hg., with a slight increase in the amount of albumin, a variable number of casts, and the same cardiac changes. Subjectively, the patient feels perfectly well, is busily engaged in teaching, and is in total ignorance of her nephritis.

Fig. 22 is from a patient who had acute dilatation of the stomach complicating pneumonia, from which she recovered to succumb to an attack of acute nephritis. The blood-pressure, which was continually low during the pneumonic infection, fell when the acute gastric dilatation

appeared, and later became higher and higher until death occured in uremia. During the attack of pneumonia, there

Fig. 22

Black dots indicate blood-pressure; open dots with solid lines, temperature; open dots with broken lines, pulse-rate.

were no urinary signs and no cardiovascular signs of nephritis.

A well marked case of acute nephritis was seen lately in the Presbyterian Hospital. The patient was an Italian boy, aged eighteen years, admitted July 19, 1913, on account of swelling of the face and swelling of the right leg and foot. Inasmuch as he had a cut on the right knee, the dispensary physician believed the edema to be secondary to an infection. The injury dated from one month ago, the swelling in the right foot and leg ten days ago, and four days ago his face became edematous and he lost his appetite.

On examination the heart outline was normal, and there was no accentuation of the second aortic. The blood-pressure was 175–120, and the urine contained many granular casts, leukocytes, and much albumin. The phenol-sulphonephthalein test gave an output of 25 per cent. in two hours. The eye-grounds were negative.

On July 15, 1913, the edema seemed more pronounced, there was a lower amount of urine, and blood-pressure was 170–100. The left border of the cardiac outline was 1 cm. outside of the midclavicular line, the right border at the right edge of the sternum, and the upper border at the third rib. The second aortic sound was accentuated, also the second sound at the apex.

From July 25, 1913, the condition improved, the blood-pressure reached 114–60 on July 31, the cardiac outline became normal and the urine contained no albumin and no casts.

This, then, is a case of acute nephritis whose pressure fell from 175–120 at the height of the attack to 114–60 when the clinical manifestations of the disease had disappeared.

Chronic Parenchymatous Nephritis.—This is a condition which, clinically, cannot be diagnosed with any degree of accuracy. Changes in the heart and blood-pressure may be entirely absent; at all events they are but very slight in comparison to those seen in cases of so-called chronic intersititial nephritis. Cases have been described which have had edema, albuminuria, and other symptoms of parenchymatous nephritis for some time but who have had low blood-pressure and no cardiac changes. When the

stage of secondary contraction occurs, the cardiovascular changes become those of interstitial nephritis, in kind, if not in degree. Others hold that hypertension in parenchymatous nephritis may be as great as in the interstitial variety. This divergent opinion is due to the edema which makes accurate blood-pressure estimations impossible.

I have lately had under my care a young Pole, aged twenty-eight years, who was admitted to the Presbyterian Hospital complaining of dyspnea and general anasarca. The edema began six months ago and increased until two weeks ago when the patient had to go to bed.

The patient was very edematous, exceedingly pale, and short of breath. The heart outline was normal; there were no accentuations and no murmurs. The blood-pressure was 129–80, there were no eye changes, the phenolsulphonephthalein test was 18 per cent. in two hours. The patient remained in the hospital from July 10, 1913, to August 27, 1913, and left considerably improved. During his stay he lost from 178½ pounds to 151 pounds, the heart did not enlarge nor did any accentuation of the heart sounds appear. The blood-pressure ranged from 129–80, to 104–55, the average being about 110.

This case illustrates low-pressure, and no cardiac changes in a long standing case of so-called parenchymatous nephritis.

Chronic Interstitial Nephritis.—In the chapter on hypertension it was stated that whenever a pressure above 150 mm. Hg. is encountered, no matter the age of the patient, a chronic nephritis should be suspected, and every means enlisted in order to diagnose this condition or to prove that it is *not* the cause of the hypertension. High blood-pressure is such a constant phenomenon in nephritis that it may be accepted as a rule that chronic interstitial nephritis is nearly always associated with hypertension. The writer has rarely seen cases with normal or hypotension, except in the terminal stages, and if they do occur, they must be looked upon as a rarity.

High blood-pressure is the earliest and most easily recognized feature of interstitial nephritis, and is a much more

constant sign than the urinary findings. It is not at all unusual to find a urine negative as to albumin and casts, but hypertension is rarely absent.

Hypertension is, therefore, to be regarded always as a manifestation of nephritis, and this view must be uppermost in the examiner's mind until he can prove that in a particular case no nephritis is present. Only until then must he look for another etiological factor. Schlayer[1] has definitely shown what an important sign of nephritis hypertension is and Janeway (Chapter V) has found that 79 per cent. of 100 patients dying with hypertension had nephritis. Only 10 per cent. of hypertension cases occur independently of nephritis, according to him.

As regards the height of the blood-pressure, there is no definite rule, it may be as high as 300 mm. Hg., but such pressures are unusual, and probably the majority will be found to be between 180 and 250 mm. Hg.

The height of the blood-pressure has little prognostic value in its relation to nephritis, but the higher the pressure the greater the danger of cerebral hemorrhage and cardiac failure.

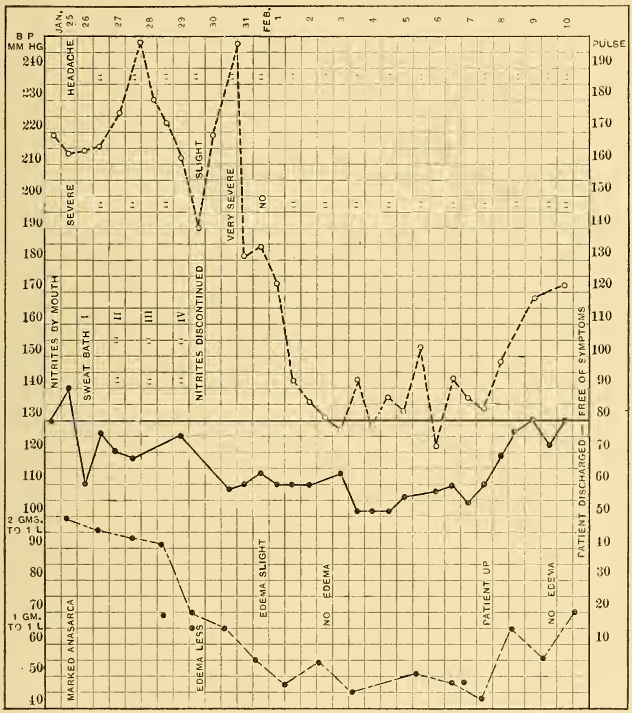
"A pressure that is seen to be going steadily up from week to week, or month to month, in spite of care on the part of the patient and the exercise of his best skill on the part of the physician, is naturally, a cause for alarm, while one that is stationary, or that under treatment is lowered to nearly normal, may make one feel more hopeful as to the immediate outlook."[2]

It must not be forgotten that hypertension, in a way is to be considered as a compensatory process on the part of Nature, and that an increased blood-pressure is required to drive the blood through the altered renal structure. The output of urine depends on pressure, for cases with high pressures excrete more urine than those with lower pressure. Finck[3] has studied the daily urine curve, and believes

[1] Münch. med. Woch., 1913, p. 63.
[2] Herrick, Osler's System, vol. vi, p. 196.
[3] La Province Méd., 1910, p. 143.

there is a constant relation between the systolic pressure and the amount of urine excreted.

Fig. 23

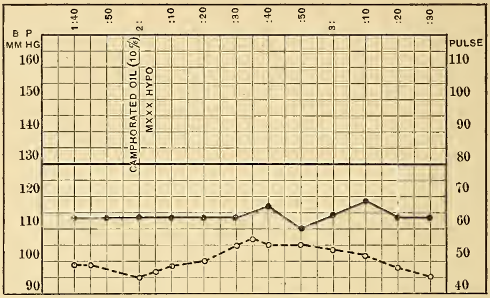


Uremia with coma. (Cook and Briggs.)

Uremia.—The blood-pressure in uremia may be high or low, but hypertension is generally seen. The two charts (Figs. 23 and 24) taken from Cook and Briggs[1] show the subjective improvement with a lowered pressure.

[1] Johns Hopkins Hospital Report, 1903, vol. xi.

Fig. 24



Uremia in chronic parenchymatous nephritis. (Cook and Briggs.)

Amyloid Kidney.—Janeway[1] reports two cases of amyloid disease with pressures of 95 and 90 mm. Hg., respectively.

The Effect of Intercurrent Infections in Nephritis.—This subject has received but scant attention although it is one which has practical importance and is worthy of close study. In pneumonia the infection seems to have little influence on the blood-pressure, the latter remaining above normal even when there is marked collapse. The writer has had lately an opportunity of observing the effect of influenza on the blood-pressure of a young woman. The pressure before the influenzal attack was 168–100 mm. Hg., and on the first day of the infection the pressure was 205–110 mm. Hg., and remained at this figure throughout the course of the disease, returning to 170–100 mm. Hg. only when the infection had run its course.

Effect of Decapsulation on Blood-pressure.—I have lately had the opportunity to study the blood-pressure in a man aged thirty-six years, who was under my care for nephritis and who was subsequently subjected to an Edebohl's operation. The blood-pressure for the first week under observation was as follows:

November 13, 1913	156–125 mm. Hg.
November 14, 1913	162–134 mm. Hg.
November 15, 1913	168–130 mm. Hg.
November 16, 1913	173–132 mm. Hg.
November 17, 1913	176–128 mm. Hg.
November 18, 1913	170–132 mm. Hg.

[1] Amer. Jour. Med. Sci., May, 1913.

The blood-pressure averaged, thereafter, between 160 and 170 mm. Hg. On December 27, 1913, the blood-pressure was 160 to 125 mm. Hg.

The man was operated upon December 31, 1913, and from this date until his death on January 17, 1914, the readings were:

January 4, 1914	126– 93 mm. Hg.
January 6, 1914	117– 88 mm. Hg.
January 7, 1914	119–110 mm. Hg.
January 8, 1914	120– 99 mm. Hg.
Jaunary 9, 1914	123–109 mm. Hg.
January 10, 1914	122– 94 mm. Hg.
January 11, 1914	125– 87 mm. Hg.
January 13, 1914	124– 79 mm. Hg.
January 14, 1914	122– 80 mm. Hg.
January 15, 1914	114– 73 mm. Hg.

As will be seen there was a marked fall in blood-pressure following the operation.

Cause of Hypertension in Nephritis.—The cause of the hypertension which is a common feature of chronic nephritis, has puzzled investigators since the time of Bright. Many theories, supported by experiments, which at first view, seemed incontrovertible, have later been shown to be merely theories. One must confess after wondering at and admiring the mental ingenuity of investigators, that despite the alluring fascination which their hypotheses have, we are as yet no nearer actual knowledge than we were fifty years ago. The subject is still in the nascent state, and what will develop therefrom one can never prophesy. In the next few pages will be found a discussion of the more important theories which have been advanced to explain the use of pressure in nephritis.

(*a*) **Mechanical Theory of Hypertension.**—Opposed to the theory of toxicity, is that of pure mechanics, which seeks to find the explanation of nephritic hypertension in a lessening of the circulation of the kidney, due to glomerular destruction. Traube, in 1856, founded this theory in the following words: "A contracted kidney diminishes the amount of fluid which is subtracted from the aortic system

to form urine, and lessens the amount of blood flowing in a unit of time from the arteries to the veins. The mean tension of the artery is thereby increased, and the heart hypertrophies from overwork due to increased peripheral resistance."[1]

Ludwig and Thiry, and others of their school, soon showed that neither increased resistance (ligation of both renal vessels) nor increase of the amount of fluid raised the blood-pressure, and Cohnheim has therefore modified this untenable view of Traube, by offering the theory that it is not the large vessels which play the important part, but rather the finer vessels which are kept in a chronic state of contraction owing to the urinary stuffs which are brought to them constantly (water, urea, sodium chlorid). Cohnheim says ligation of both vessels proves nothing, as the organism can well take care of this increased amount of blood, but in nephritis there is a disproportion between the amount of blood and the contracted vessels.

Alwens[2] using a reconstructed technique, believes that the cause of the hypertension is *purely* mechanical, and thinks he had demonstrated that there is increased intrarenal pressure, which involves the entire arterial system. Chemical and nervous influences are absolutely disclaimed.

The question of the mechanical theory seems to have received its quietus from Senator.[3] The latter produced embolism of the smallest renal vessels by injecting liquid paraffin into both kidneys. In no case was there a rise of the carotid blood-pressure, and Senator says "Herewith is the so-called physical theory of hypertension and hypertrophy dismissed once for all," although Janeway[4] found, after reducing the kidney substance in dogs that there was a rise in blood-pressure which gradually fell before death (terminal period).

[1] For full literary references, see article by Janeway on this theme, Amer. Jour. Med. Sci., May, 1913.

[2] Deutsch. Arch. für klin. Med., 1909–10, xcviii, p. 137.

[3] Ztsch. für klin. Med., 1911, lxxii, p. 189.

[4] Proceedings Soc. Exper. Biology and Med., 1909, vi, p. 109.

	Average.
Before operation, fifteen days	106
After operation, twenty-one days	127
Terminal period, 11 days	83

Another dog which lived gave the following results:	Average.
Before operation, forty-five days	90
After operation, first period, forty-three days	111
After operation, second period, twenty-six days	121
After operation, third period, thirty-one days	125

(*b*) **The Effect of Kidney Extracts on Blood-pressure.**[1]—In 1898, Tigerstedt and Bergmann[2] published a very extensive series of experiments, which appeared to demonstrate the presence of a pressor substance in extracts of rabbits' kidneys. The substance was found almost exclusively in the renal cortex and was present not at all or only to a slight extent in the medulla. It could be extracted from the fresh organ by salt solution, by alcohol, by fresh blood, and to a less extent by cold water. Boiling seemed to destroy its action. The substance was non-dialyzable and the investigators, therefore, concluded it could not be any of the urinary salts. They regarded it as an internal secretion of the kidney which passes normally into the circulation and named it "*renin.*" The rise of pressure which followed its injection varied from a few millimeters to 25 to 35 mm. Hg., due, they thought, to its action on the peripheral nerve centres or on the spinal cord. Very small amounts caused as much effect as larger doses, and repeated injections produced each an effect as great as the first injection.

In order to demonstrate the passage of this supposed pressor substance into the circulation, Tigerstedt and Bergman injected blood from the renal vein into the vessels of rabbits which had suffered double nephrectomy. A moderate rise followed (18 mm. Hg.) These last experiments were repeated by Lewandowsky[3] and although a rise of pressure was observed, he obtained similar transient pressor effects in his controls by injecting blood from the

[1] The author is indebted to Pearce (Arch. Int. Med., 1908, ii, p. 77) for much of the material of this section.

[2] Skand. Arch. für Physiol., 1898, viii, p. 223.

[3] Ztsch. für klin. Med., 1899, xxxvi., p. 535.

general venous (jugular vein) and also arterial systems. Lewandowsky concludes there is some pressor substance in the blood, but he does not share Tigerstedt and Bergman's theory that it is an internal secretion of the kidney. Subsequent investigators have repeated Tigerstedt and Bergman's work with indifferent success.

Pearce has subjected the research to a critical review, and points out the striking fact that the result obtained seems to depend on whether or not the kidney extract was injected into an animal of the same species. His own experiments offer no support to the theory that a pressor substance or substances exist in the normal kidney. It was found that the injection into the rabbit of extract of either dog or rabbit kidney caused a slight rise in pressure but that similar injections into the dog caused a depressor effect, which, when dog's kidney was used, was very decided. It is evident, therefore, that the pressor substance of the kidney of any given species has no constant pressor effect for animals of other species, as is the case with adrenalin. Almost any substance, blood-serum, defribrinated blood, urine, extracts of the liver of both rabbit and dog, urea, sodium chlorid and Locke's solution when injected into the rabbit's circulation in doses of from 1 to 3 c.c., caused a slight transient rise in pressure. Pearce concludes that, since there is a rise in pressure by such a variety of substances, the effect of the injection is largely mechanical.

Bengel and Strauss[1] apparently without being aware of Pearce's work, have reached the conclusion that there is in the kidney a pressor substance which is different from adrenalin in its action, being influenced by section of the vagus or sympathetic, or by the destruction of the spinal cord, or even by extirpation of the kidneys, adrenals, and liver, and they assume that its action is solely on the musculature of the arteries. They confirm the work of Tigerstedt and Bergmann, and believe that "*renin*" must have some bearing on the rise of blood-pressure seen in cases of contracted kidneys, although they insist that their work has shown nothing to prove this assumption.

[1] Deutsch. Arch. für klin. Med., 1909, xcvi, p. 476.

(*c*) **Adrenalin Theory.**—Owing to the frequent occurrence of circumscribed or diffuse hyperplasia of the adrenals in cases of contracted kidney, Vaquez and Aubertin[1] suppose that there is some intimate relationship between the nephritic process and this hyperplasia, which they believe is synonymous with hyperadrenalism. The French school has been most actively engaged in pointing out this striking occurrence, especially in cases where the disease has had a more or less chronic course. Pearce[2] has given the matter considerable attention and finds very definite and fairly constant changes in the adrenals in general arteriosclerosis from whatever cause, interstitial nephritis, parenchymatous nephritis, and general arteriosclerosis with no evident renal lesion. He believes that the alterations described by Vaquez and his followers are not peculiar to chronic interstitial nephritis, but are found in all conditions with advanced arteriosclerosis. Pearce concludes that a correlation existing between the diseased kidney and the adrenals and having an influence on the vascular system is doubtful.

In May of 1907, Schur and Wiesel[3] discovered that the serum of a nephritic caused mydriasis of the enucleated frog's eye (Ehrmann's Test) and later were able to prove chemically that the blood-serum contained adrenalin, by applying the fertic chlorid reaction. On these findings they assumed that in nephritis, certain substances were retained which acted as a secretary excitant to the adrenals, and that the outpouring of increased amounts of adrenalin explained the associated hypertension. The Ehrmann test was positive in practically all cases of nephritis, and was also present in animals with double nephrectomy, and, to a less extent, in animals with experimental nephritis.

Schlayer[4] has repeated the work of Schur and Wiesel, using the method of Meyer. Meyer, be it remembered,[5]

[1] Bull. Soc. Méd. d'hôp. de Paris, 1905, xxii, p. 705.
[2] Arch. Int. Med., 1908, ii, p. 77.
[3] Wien. klin. Woch., 1907, p. 1202.
[4] Deutsch. med. Woch., 1907, p. 1897.
[5] Ztsch. für Biol., 1906, p. 352.

found that arterial walls (vessel strips) could be preserved for a long while, and could be used to study the action of various chemicals on smooth muscle fibre, and on the nerve endings in the vessel wall. One end of the vessel strip is fixed, and to the other end a ligature is fastened, armed with a point which writes on the smoked drum of a kymographion. He found that adrenalin in dilutions of 1,000,000 to 100,000,000 could be detected by this method (0.000015 mg. in 15 c.c. Ringer's solution). Using the Meyer method, Schlayer observed a strong contraction of the vessel wall with normal human blood-serum, but not with beef serum. Further studies revealed to him a decided analogy between adrenalin and this unknown pressor substance.

Turning his attention to nephritis, he held the view, that if there is really an adrenalinemia in nephritis, it must run quantitatively, *pari passu,* with the degree of arterial pressure. Eight cases were studied (pressure 190–260 mm. Hg.), and in all, 26 observations were made. His results are given in the accompanying table:

No.	Type of nephritis.	Blood-pressure.	Contraction of the artery in mm.	
			Nephritic serum.	Normal serum.
4	Primary interstitial	195	32	25
13	Lead poisoning	110	50	23
10	Lead poisoning	212	25	33
17	Lead poisoning	208	12	17
5	Primary interstitial	260	18	34
6	Secondary interstitial	190	10	29
9	Secondary interstitial	200	31	35
20	Secondary interstitial	195	26	28
15	Secondary interstitial	190	11	23
11	Secondary interstitial	209	15	22

It will be seen that of the 26 experiments, the nephritic serum caused a stronger contraction than normal serum in but two. The other cases showed lower pressor powers of the nephritics than of the control sera. That it was not owing to the dilution caused by the hydremia,

was proved by using the dried residue, but with the same result. Schlayer concludes that there is no evidence to prove the relationship between hypertension of the adrenalin content of the blood serum. Later authors, among whom may be mentioned Fränkel,[1] Aschoff and Cohn,[2] Obendorfer[3] have also been unable to confirm Schur and Wiesel's findings. Ingier and Schmorl[4] have studied the epinephin content of the adrenal's certain diseases and have demonstrated larger amounts in chronic nephritis, true and arteriosclerotic contracted kidneys, than in other conditions.

That there is a pressor substance in the blood-serum of nephritics there can be no doubt, the only question which seems vital at the present time is whether that substance is epinephrin.

Stewart[5] insists that no single method should be used as evidence of adrenalinemia, and he suggests, in order to diminish the chance of error in testing blood for adrenalin, to use a combination of bloodvessel and intestines, as adrenalin causes contraction of the former and inhibits contraction of the latter. He thinks there is no detectable amount of adrenalin in normal blood, and in a second paper[6] he found no indication of adrenalin in pathological sera.

After making comparative studies with the vessel strip method, the Läwen-Trendelenburg method and the intestinal method, O'Connor[7] finds that while there is a pressor substance in the blood, it is certainly *not* adrenalin. Furthermore, he discovered that this adrenalin-like body was not found in natural blood, but arose during the process of clotting. In rabbits, adrenalin could be detected in the blood of the suprarenal vein where it was present in dilutions of 1 to 1,000,000 or 1 to 5,000,000. Section of the splanchnics markedly diminished the amount of adrenalin.[8]

[1] Arch. für exp. Path. and Pharmakol., 1909, Band lx.
[2] Verhandl. d. path. Gesellsch., 1908, p. 131.
[3] Ibid., 1909, p. 273.
[4] Deutsch. Arch. für klin. Med., 1911, Band 104.
[5] Jour. Exp. Med., 1911, xiv, p. 377.
[6] Jour. Exp. Med., 1912, xv, p. 547.
[7] Arch. für exp. Path. and Pharmakol., 1911-12, xlvii, p. 195.
[8] Ibid., 1912, xlviii, p. 383.

Janeway and Park[1] working with a technique which embraces a modification of the Meyer method, by using carotid and coronary arteries as controls, have found there is no evidence that epinephrin exists in the circulating blood in amounts sufficient to produce its physiologic effects. An exception to this is the suprarenal vein which always contains it. The quantity of epinephin required to cause minimal hypertension is at least ten to twenty times the amount secreted by the adrenal glands.[2]

Despite the fascination which the adrenalin theory seems to have, one is forced to admit that there is no just ground for assuming that adrenalin is the substance which raises the blood-pressure in nephritis. Until Janeway and Park's work appeared, many were inclined to the view that the pressor substance was an epinephrin-like body, but even this last straw is washed away in the flood of evidence offered in their research. The substance, whatever it is, will doubtless prove to be a very familiar body, in all probability a well-known component of the protein molecule. This has been hinted at in the work of Lindemann[3] who has produced renal changes by the injection of serum from a nephritic animal into a normal animal, and is suggested also in the work of Pearce.[4]

Hess and Wiesel[5] have shown that injections of adrenalin will prevent the death of rabbits poisoned with uranium nitrate.

(*d*) **Hypertension and Blood-sugar.**—Working on the knowledge that adrenalin can cause diabetes (adrenalin diabetes—von Noorden) some have assumed that if it is an abnormal adrenalinemia which causes the hypertension, then hypertension must be associated with hyperglycemia and glycosuria. Neubauer[6] has found in a number of uncomplicated cases of nephritis, a hyperglycemia, so that there was a

[1] Jour. Exp. Med., 1912, xvi, p. 541.
[2] Haskins and McClure, Arch. Int. Med. 1912, x, p. 343
[3] Ann. de l'Inst. Pasteur., 1900, xiii, p. 49.
[4] Arch. Int. Med., 1908, ii, p. 77.
[5] Wien. klin. Woch., 1913, p. 317.
[6] Biochem. Ztsch., 1910, xxv, p. 284.

definite relation between the blood-sugar content and the high blood-pressure, the greater the hyperglycemia the greater the hypertension. These observations have been emphatically denied by subsequent workers, notably Stilling,[1] Weiland[2] and Tachau.[3]

Hagelberg[4] endorses, however, in no uncertain manner Neubauer's observations, finding not only a hyperglycemia but an alimentary hyperglycemia in nephritis and believes that in arteriosclerosis and in nephritis there is an increased adrenalinemia.

Orthostatic Albuminuria.—The constant low pressure which is seen in postural albuminuria serves in the majority of cases to distinguish it from true nephritis.

Double Nephrectomy.—Extirpation of both kidneys is without effect on blood-pressure.[5] The explanation offered is that normally there is a hypertensive substance in the blood which is eliminated in the urine. The double nephrectomy causes a retention of such a substance and, hence, hypertension. Physical factors play no role as there is less work on the left ventricle owing to the elimination of the glomerular circulation, and the blood-pressure, theoretically at, least should be lowered.

[1] Archiv. für exp. Path. u. Pharmakol., 1911, lxvi, p. 238.
[2] Zentralbl. für d. ges. Phys. u. Path. d. Stoffw., 1910, p. 13.
[3] Deutsch. Arch. für klin. Med., 1911, p. 102.
[4] Berl. klin. Woch., 1912, p. 1877.
[5] Mosler Ztsch. für klin. Med., 1912, lxxiv, p. 297.

CHAPTER VIII.

ACUTE AND CHRONIC INFECTIONS INCLUDING CERTAIN INTOXICATIONS.

Some years ago it was prophesied that but very little of importance would be obtained from a study of blood-pressure in acute diseases. We shall see that such a forecast meets refutal in two notable instances, typhoid fever and pneumonia, so it would be hazardous to say that, because in the vast majority of acute fevers nothing as yet has been brought forward, the future will not reveal some valuable diagnostic or prognostic phenomena. The fact remains, however, that many acute diseases, are, as a rule, uninteresting from the stand-point of blood-pressure, but the probable explanation of this is that they have not been so closely studied as have pneumonia, typhoid fever and cerebrospinal meningitis, for instance.

ACUTE INFECTIONS.

Typhoid Fever.—Typhoid fever, owing to the danger of fatal complications, offers opportunity for the judicious use of the sphygmomanometer. In the large cities, the disease has become so rare, that it does not possess the same significance as it formerly did, but the complications are nevertheless ubiquitous, and should be as closely watched for as before. All authors are agreed that typhoid fever is associated with arterial hypotension, and the figures of Crile[1] are of interest in showing the pressure during the course of the infection. 115 cases were studied with the Riva-Rocci instrument and of these:

The highest pressure was 138 mm. Hg.
The lowest pressure was 74 mm. Hg.
The mean pressure was 104 mm. Hg.

[1] Jour. Amer. Med. Assoc., 1903, xl, p. 1292.

During the course of the disease the mean pressure was found to be as follows:

First week 115 mm. Hg.
Second week 106 mm. Hg.
Third week 102 mm. Hg.
Fourth week 96 mm. Hg.
Fifth week 98 mm. Hg.

Cook and Briggs[1] claim that there is no pathological condition apart from shock, in which careful blood-pressure readings are prognostically more significant. They point out that the arterial hypotension rises *pari passu* with the degree of the toxemia, a fact beautifully illustrated by Crile's figures. Blood-pressure estimations should be recorded as frequently as the temperature respiration and pulse rate, and the readings should be made a part of the nurse's duties as well as the registration of these other clinical data. The technique is comparatively simple and it would be a great step forward, clinically and scientifically, to have this suggestion become a realized fact. It is obvious that blood-pressure cannot be taken by the physician as often as the temperature pulse and respiration are recorded, and hence this office must fall to the nurse.

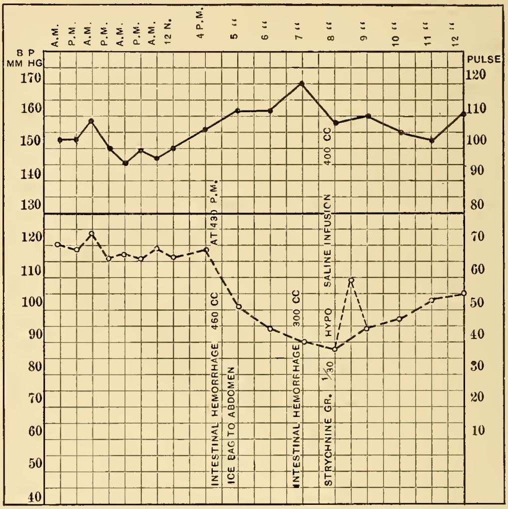
The significance of blood-pressure in typhoid and indeed in many conditions, can only be appreciated when one has had previous daily and perhaps, hourly records with which to compare deviations. Isolated readings are valueless, Gumprecht[2] has shown that individual quotidian variations occur as they do in health.

Hemorrhage.—It is often essential and sometimes difficult, to distinguish between intestinal hemorrhage with pain, and peritoneal irritation, if not actual peritonitis, and it is here that blood-pressure estimations will be found of vital importance. Hemorrhage is generally accompanied by a fall in pressure as is well shown by the chart reproduced from Cook and Briggs, although this is not a constant feature. The author has lately seen a case of fatal hemor-

[1] Johns Hopkins Hospital Reports, 1903, xi, p. 502.
[2] Ztsch. für klin. Med., 1900, xxxix, p. 377.

rhage, but with no fall of blood-pressure below the former limit of hypotension (98 mm. Hg.).

Fig. 25



Typhoid fever—intestinal perforation. (Cook and Briggs.)

Perforation.—Just as hemorrhage is usually followed by a fall in pressure, perforation is generally associated with a sharp rise. Lately a case of perforation was observed which fell from 124 mm. Hg. to 110 mm. Hg., and remained there until operation was performed. Hypotension of itself is, therefore, not a safe indication that there is no perforation.

Crile quotes five cases of perforation. The first showed a rise from 116 mm. to 190 mm. in four hours. The second case was admitted with general peritonitis and a pressure of 105, later falling, with general improvement, to 80. On the eighth day, peritonitis set in and the pressure rose from 84 to 100 mm. Hg. In a third case with slowly forming perforation the blood-pressure rose from 116 to 165 mm. in two hours, but with but little increase in pulse rate.

In cases 4 and 5 the pressure was 165 and 208 respectively.
Twenty of Crile's cases of acute peritonitis gave the following pressures:

Highest	208
Lowest	156
Mean.	166

Fig. 26

Typhoid fever—intestinal hemorrhages. (Cook and Briggs.)

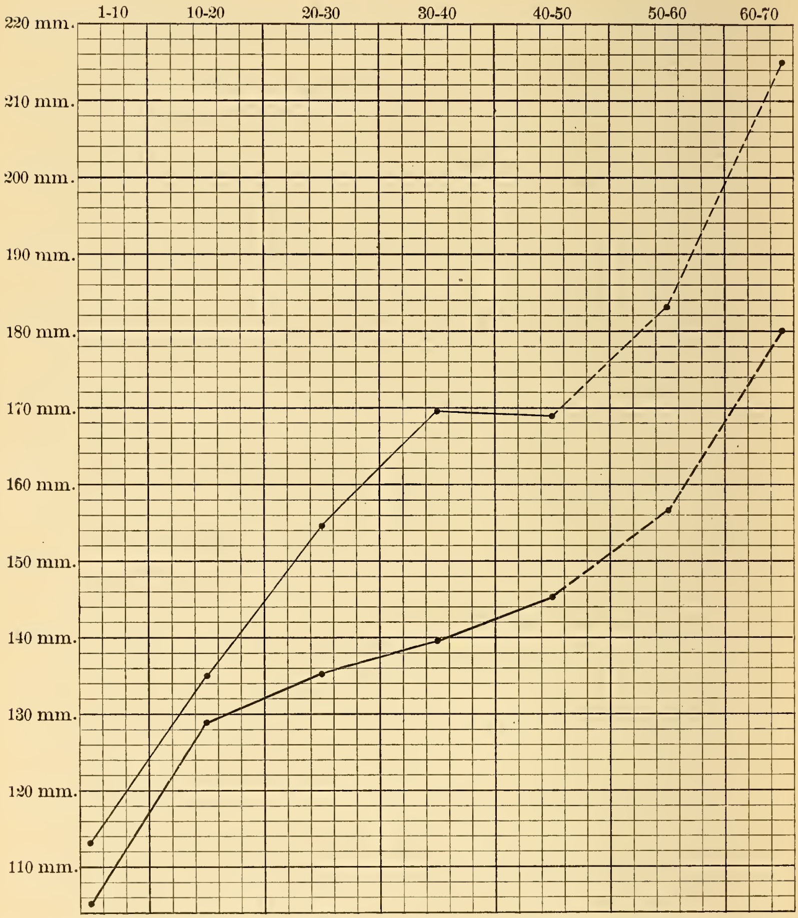
Cook and Briggs report similar experiences and state that "A sharp and well maintained rise in blood-pressure in typhoid fever, from a previously known low level, is a valuable sign of intestinal perforation, and is probably of more constant value than the notoriously uncertain increase in the leukocyte count." They question the negative value of a rise in systolic pressure in doubtful cases with suggestive abdominal signs.

Late Effect of Typhoid Fever on the Heart and Vessels.—Thayer[1] examined 183 patients who had had typhoid fever

[1] Amer. Jour. Med. Sci., 1904, cxxvii, p. 391.

from one month to thirteen years previously, and his results are well shown in the accompanying table.

FIG. 27



OLD TYPHOIDS	113.2 (4 CASES)	134.9 (28 CASES)	152.2 (32 CASES)	169.9 (24 CASES)	168.7 (8 CASES)	183.5 (2 CASES)	215 (1 CASE)
HEALTHY INDIVIDUALS	105.5 (32 CASES)	128.3 (62 CASES)	135.1 (52 CASES)	139.8 (19 CASES)	145.2 (11 CASES)	156.2 (4 CASES)	180 (1 CASE)

Showing the averages of the systolic blood-pressure in old typhoids and in normal individuals from whom all cases with a history of serious infectious diseases or alcoholic habits have been excluded. Upper tracing, old typhoids; lower tracing, healthy individuals.

"The highest record of blood-pressure among the cases in healthy individuals was 180, and that in a woman aged sixty years, while among the old typhoids there were 27 cases in which the pressure was above 180, 10 showing a record of 200 or above." Thayer's observation would point to a hitherto unsuspected frequency of post-typhoidal cardiovascular changes.

Pneumonia.—There is a marked difference of opinion as to the blood-pressure in pneumonia. Kaufmann and deBarry[1] believe the pressure is low, and their view is shared by Norris, Gibson,[2] Cowan[3] while others, notably Cook and Briggs, Gilbert, Castaigne[4] hold that the disease is hypertensive. Gibson says that the pressure is lowest at the time of the crisis or immediately after, and rises during the next few days.

During the winter of 1910–11 the author had opportunity to observe 20 cases of pneumonia, admitted to the service of J. H. Musser in the Presbyterian Hospital, and also five cases on his service in the University Hospital. I have tabulated these results:

Between 80 and 90 mm. Hg.	1
Between 90 and 100 mm. Hg.	1
Between 110 and 120 mm. Hg.	11
Between 120 and 130 mm. Hg.	3
Between 130 and 140 mm. Hg.	2
Between 140 and 150 mm. Hg.	3
Between 160 and 170 mm. Hg.	2
Between 180 and 190 mm. Hg.	1
Between 210 and 220 mm. Hg.	1

The lowest pressure was 83 and the highest 212 mm. Hg.

There are so many factors which must enter into the question before an opinion can be given, namely the question of age, condition of heart and bloodvessels, condition of kidneys, all of which modify the picture. The day of the disease may also play some role, but it must not be forgotten that quotidian variations are also manifest in this

[1] Berlin. klin. Woch., 1888, p. 557.

[2] Edin. med. Jour., 1910, p. 31.

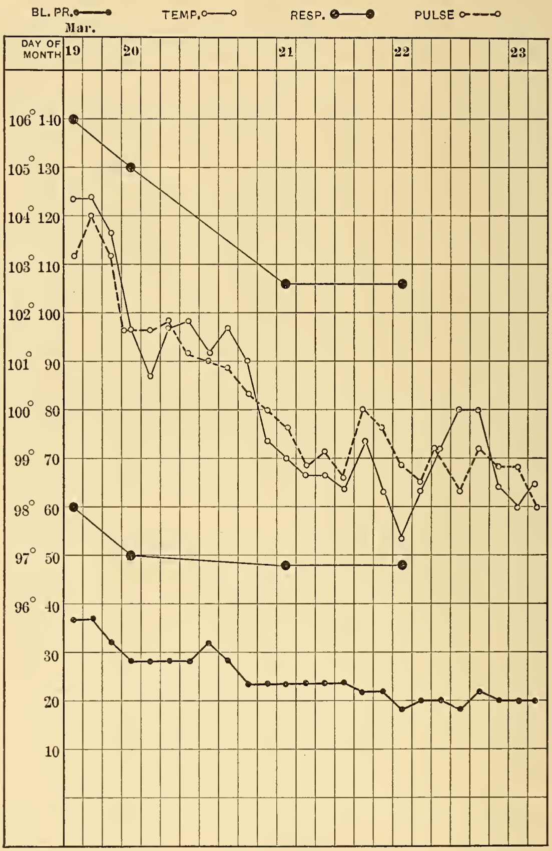
[3] Practioner, 1904, lxxiii, 218.

[4] Sean. et mém. de. la Soc. de la Biol., 1899, i, 633.

9

disease and in one case seen in the University Hospital the changes were as great as 17 to 20 mm. Hg.

Fig. 28



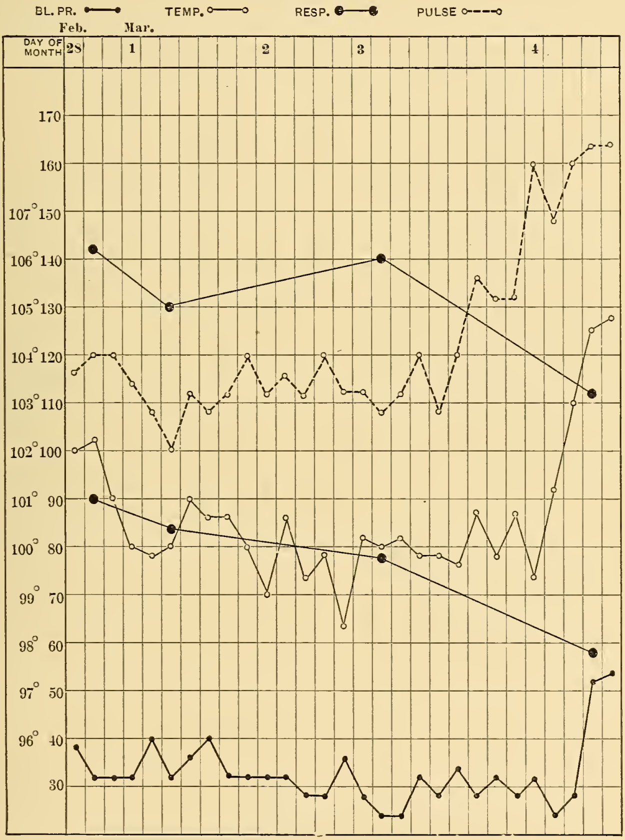
Blood-pressure above pulse rate. Recovery. Heavy black dots and lines, blood-pressure; interrupted open dots, temperature; open continuous dots, pulse rate; closed black dots, respiration.

Hensen[1] has noted a rise of pressure in dyspneic states, and Janeway recalls the influence which individual reaction

[1] Archiv. f. kl. Med., 1900, lvii, p. 436.

amount of tissue involved, dyspnea, cyanosis, and many other factors exert on the blood-pressure.

FIG. 29

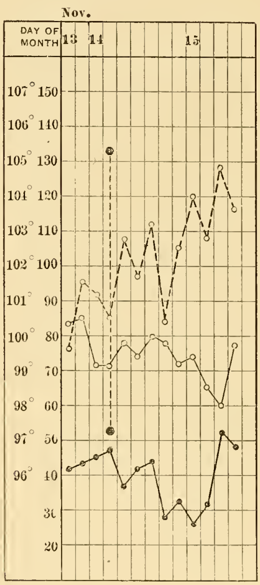


Blood-pressure below pulse rate. Death.

The value of observing the ratio of blood-pressure to the pulse rate has been pointed out by Gibson, Gordon, and others. It is claimed that when the blood-pressure expressed in millimeters of mercury remains above the pulse rate

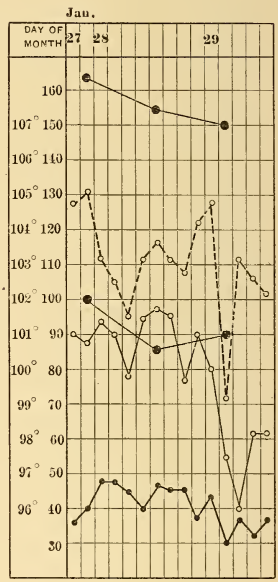
expressed in beats per minute, the case has a favorable prognosis. This purely empirical observation has been thought so highly of that some claim never to have seen a fatal case when the blood-pressure and pulse ratio is thus preserved (see Fig. 28), and rarely a favorable outcome when the pulse rate rises above the blood-pressure (see Fig. 29).

Fig. 30



Blood-pressure above pulse rate. Death.

Fig. 31



Blood-pressure above pulse rate. Death.

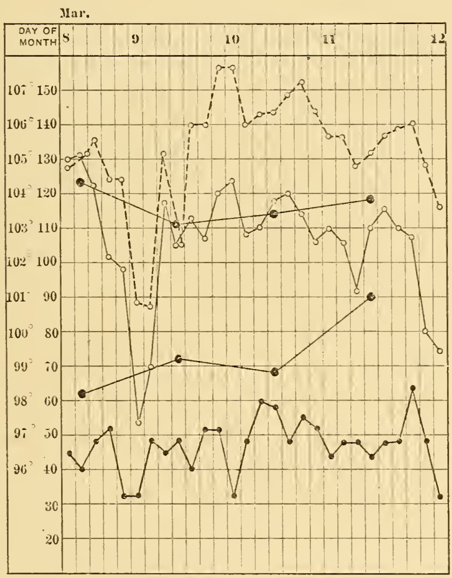
In 1911 the author[1] reported some work undertaken along these lines. 22 cases were studied in the Presbyterian Hospital, and although it is regretable that blood-pressure readings were not made more frequently, some interesting facts were learned. 18 of these cases correspond exactly with Gibson and Gordon's findings; in 12 there was a favorable outcome, the blood-pressure being maintained

[1] Therap. Gaz., July 15, 1911.

above the pulse rate, and in 6 the blood-pressure fell below the pulse rate and death ensued.

That the rule of Gibson is, however, too dogmatic and inflexible to permit of general acceptance, is shown in Figs. 30, 31, 32, and 33.

FIG. 32



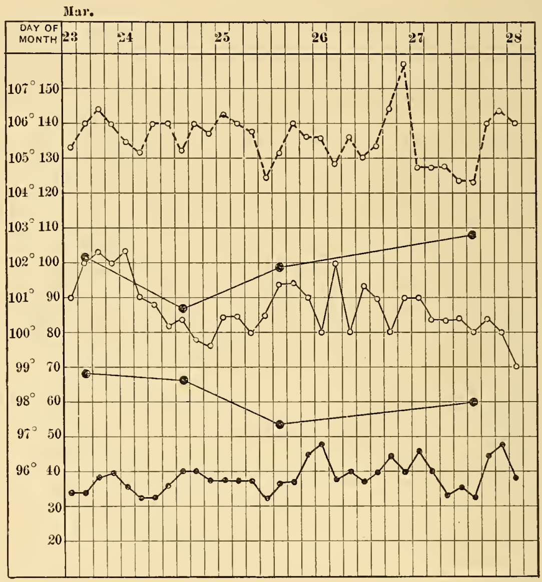
Blood-pressure below pulse rate. Recovery.

Figs. 30 and 31 are of patients who had a higher blood-pressure than pulse rate, but who nevertheless died, and Figs. 32 and 33 show a favorable outcome when the pulse rate was continuously higher then the blood-pressure.

While admitting the clinical usefulness of observing the blood-pressure, pulse rate ratio, the author does not believe that a fatal result always accompanies a blood-pressure lower than the pulse rate nor does he regard a blood-pressure higher than the pulse rate as a sign of ultimate recovery. It must not be forgotten that nephritis or some conditions raising blood-pressure of itself, may be

present in the subject suffering from pneumonia, and hence raise that individual pressure far above normal despite the influence of the infection. Such individuals are in just as much danger from collapse as those with much lower pressure, and treatment is as urgently indicated.

Fig. 33



Blood-pressure below pulse rate. Recovery.

Stimulation is rarely required when the blood-pressure remains above the pulse rate, and this ratio is most useful in guiding the administration of drugs, the latter generally being necessary when blood-pressure falls below the pulse rate.

Epidemic Cerebrospinal Meningitis.—Increased intracranial pressure is always associated with cerebrospinal meningitis, and on account of the intricate relationship existing between the former and the elevation of blood-pressure, which we have learned from Cushing to be a fundamental truth, we should expect to find the pressure

elevated in meningitis of the epidemic variety. Robinson[1] has made the most comprehensive study of this question which has yet appeared.

Observations were made in 26 cases of meningitis and from these, 336 blood-pressure estimations were charted. The effect on blood-pressure of removal of fluid by lumbar puncture was studied 38 times in these 26 cases and seven times in other conditions. The Stanton instrument with a 10 cm. arm band was used, and the systolic pressure was determined by palpation.

The tension of the cerebrospinal fluid as measured by a manometer was as follows:

Above 500 mm.	3 times
400 to 500 mm.	5 times
300 to 400 mm.	4 times
200 to 300 mm.	2 times
150 to 200 mm.	2 times

The normal pressure being about 120 mm. there is evidence in the above table of a constant increase in intracranial pressure. There was no constant relation between blood-pressure and the heightened intracranial pressure, although there was at times an increased blood-pressure, which makes meningitis an exception to the rule that "in infectious diseases the pressure is lowered."

As a rule heightened blood-pressure is seen in the early acute stage when exacerbations of symptoms occur, late in the disease, or when the condition becomes chronic. It is higher when the symptoms are severe and low during convalescence.

Robinson points out "the lack of uniformity in the blood-pressure curves, and the great irregularity of the individual curves, the highest blood-pressures were encountered in two stages of the disease, early, with the severe symptoms of onset, and later, with the pre-terminal symptoms, before the final failure of the circulation appeared." He finds a relationship between the severity of the symptoms and blood-pressure, the pressure increasing with increase of symptoms,

[1] Archives Int. Med., 1910, v, p. 482.

namely, fever, headache, delirium, rigidity of the neck, and ocular disturbances.

The writer derived great assistance recently from the sphygmomanometer in a suspected case of meningitis. The patient was a negro, who was admitted to the Presbyterian Hospital with a diagnosis of typhoid fever. The blood-pressure was 110 mm. Hg. and a careful study led ultimately to the diagnosis of paratyphoid fever. A few days after admission, the patient began to show signs of meningitis, *i. e.*, rigidity of the neck, spasticity of the limbs and cerebral symptoms. The blood-pressure, however, did not rise, and the lumbar puncture, apart from revealing increased pressure of the cerebrospinal fluid, showed nothing to indicate the presence of a meningitis. Following the puncture, the patient improved, and the meningeal symptoms gradually disappeared. Future work may show that in low blood-pressure we have a means of helping to distinguish meningeal irritation from true meningitis.

The effect on blood-pressure of lumbar puncture is not constant, although it is frequently lower after than before, and the hypertension which is frequently seen in epidemic cerebrospinal meningitis is probably not due to the increased intracranial tension. Muscular movements such as those accompanying delirium may play a role, or there may be reflex stimulation of the blood-pressure raising mechanism of the body, by irritation of the cerebral nervous mechanism as by pain. Internal hydrocephalus or pyohydrocephalus may also be causes of the increased blood-pressure.

It is interesting to see such an inconstant fall of arterial pressure after lumbar puncture, as we know that the puncture generally brings about a considerable drop in pressure (see Lumbar Puncture).

Diphtheria.—Rolleston[1] has made most careful observations on 179 cases of diphtheria, using the palpatory method of determining systolic pressure. His conclusions which are affixed to a classic article, containing good bibliography, are:

1. In a series of 179 cases of diphtheria, the blood-pressure

[1] Brit. Jour. Chil. Dis., 1911, viii, p. 433.

was found to be subnormal in 63 patients, or 35.1 per cent., the extent and duration of the depression having, as a rule, a direct relation to the severity of the faucial attack.

2. In the great majority, the highest readings are found in the first, and the lowest in the second week of disease. The normal tension is usually reëstablished by the seventh week.

3. In a large proportion of convalescent cases, either the readings in the recumbent and erect positions are the same, or the recumbent is higher than the vertical record until convalescence was firmly established.

4. In laryngeal cases, disproportionately high readings are obtained, especially when the dyspnea is sufficiently severe to require operation. Relief of the obstruction by tracheotomy is followed by an immediate and steep fall of blood-pressure (20 to 40 mm.).

5. The blood-pressure shows little tendency to be affected by the early serum phenomena, but during the late febrile syndrome it may be raised.

6. Albuminuria is accompanied either by a fall or by no change in the blood-pressure, except in cases of uremia in which there is hypertension.

7. In early paralysis, the blood-pressure tends to fall. In late paralysis, even when extensive, it is usually not affected.

8. Sphygmomanometry in diphtheria, as in other acute diseases, though of considerable theoretical interest, has little practical significance.

9. Adrenalin therapy in diphtheria may favorably influence the other symptoms of suprarenal insufficiency without affecting the blood-pressure.

In the fatal cases, there is little prognostic importance to be attached to blood-pressure estimations. In the majority of cases the pressure was variable in its manifestations, in no cases did death take place unexpectedly, the characteristic features of malignant diphtheria indicating a lethal outcome before there was any lowering of blood-pressure.

Anaphylaxis.—The distressing but happily infrequent phenomena of "anaphylactic shock," are now supposed to be due to spasm of the unstriated muscles of the finer divisions of the bronchial tree, death being due to strangulation.

This condition is produced by peripheral action and seems to be in no wise of central origin. There is with the phenomenon of anaphylaxis a rise in blood-pressure and later a fall. Atropin brings about relaxation of the over-distended lung, and if given before the serum it may prevent the fatal lung distension.[1]

Pearce and Eisenbrey[2] have studied the question of anaphylactic "shock" in dogs, and describe an abrupt fall of blood-pressure, averaging 50 to 70 mm. of mercury. Respiration is unaffected apart from disturbances due to anemia of the medulla, arising from the low blood-pressure. The low blood-pressure and the associated decreased peripheral circulation are accompanied by congestion of the veins of the splanchnic area, flooding of the splanchnic sea. The authors confirm the findings of Auer and Lewis and show conclusively that the low pressure is caused by influences on the peripheral nerve endings of the vasomotor mechanism. The "shock" is accompanied by a sudden persistent fall in blood-pressure and by symptoms due to cerebral anemia.

Scarlet Fever.—A study of the systolic pressure in cases of scarlet fever shows the pressure to be subnormal in 25 per cent. of the cases, the hypotension being in direct relation to the severity of the onset of the disease. The highest of these low tensions are found in the first week, but there is a predominence of the lowest, also in this week, although the majority of the lowest readings are obtained in the second week. Normal pressure is not attained until the fourth week. In convalescence the tension is lower than in the acute stages.

Complications have little effect on blood-pressure. In only a small proportion of the cases showing signs of renal insufficiency is there a rise in blood-pressure, and even in these cases the hypertension was never extreme or of long duration. Sphygmomanometry has here, as in diphtheria, but little prognostic value. The early fatal cases do, it is true, show a marked fall in pressure than

[1] Auer and Lewis, Jour. Amer. Med. Assoc., 1909, liii, p. 458.
[2] Jour. Infec. Diseases, 1910, vii, p. 565.

do the fatal cases of diphtheria, but this has little significance. Blood-pressure readings should be made in cases of postscarlatinal nephritis, as such may show the extent of the renal lesion. This hypertension and the pronounced low tensions occasionally encountered, are indications for treatment.

Cholera.—An interesting research on blood-pressure during the cholera epidemic in St. Petersburg, (1909–10), has been made by Lang.[1] He has found in the algid state, a lowering of systolic pressure and a rise in diastolic, this being due to a small volume of blood (loss of water) with a consequent narrowing of the lumen of the vessels. Salt solution in quantities of two liters causes the blood-pressure to approach more nearly the normal, on account, probably, of restoration of the total volume of liquid in the blood-vessels. When too much salt solution is injected (intravenous), that is, when more is administered than fluid lost, blood-pressure, both maximal and minimal, rises above normal.

After the algid state, there is a rise of blood-pressure for the first few days.

Federn[2] takes an entirely different view and says, in cholera, "the blood-pressure is 140 mm. and perhaps higher, as I have seen no patient in the algid state,"

Malaria.—Federn[3] made observations on three cases, and finding the pressure so characteristically raised, he concluded this must be the rule. The pressure is raised only in the febrile stage, during the chill and fastigium of fever, and drops when sweating begins.

Norris,[4] on the other hand, finds nothing of importance to be derived from blood-pressure observations in malaria.

Other Infections.—**Acute Articular Rheumatism.**—Norris found no change of consequence, but Potain[5] considered it a hypertensive condition.

[1] Deutsch. Arch. für klin. Med., 1912, cviii, p. 236.

[2] Verhandl. d. Kongresses für inn. Med., 1904, xxi, p. 582. [3] Loc. cit.

[4] Amer. Jour. Med. Sci., 1903, cxxv, p. 880.

[5] La pression arterielle de l'homme a l'état normal et pathologique, Paris, 1902.

Influenza.—Hypotension is the rule in influenza.

Paratyphoid Fever.—Three cases of paratyphoid fever, whose blood contained specific agglutinins for the alpha strain, have been under my care lately, and in each there was hypotension.

II. CHRONIC INFECTIONS AND INTOXICATIONS.

Tuberculosis.—Schnitter[1] has given a concise table of the blood-pressure in the various stages of tuberculosis. He has divided his cases into the three degrees suggested by Turban, namely:

First Degree.—Diseases of slight severity, affecting at most one lobe or two half lobes.

Second Degree.—Disease of slight severity, more extensive than first, but affecting at most two lobes, or severe and affecting at most one lobe.

Third Degree.—All cases of greater extent and severity than second.

Systolic pressure.	Tub. pul. First degree.	Tub. pul. Second degree.	Tub. pul. Third degree.	Only afebrile tub. pul.
60 to 100 mm. Hg.	35	35	62	26
100 to 115 mm. Hg.	39	53	29	37
115 to 130 mm. Hg.	16	4	4.5	26
Above 130 mm. Hg.	10	8	4.5	11

It will be seen that the more severe the tuberculosis the more often will low pressure be observed and the greater will be the hypotension. Of the 100 cases of tuberculosis tabulated above, 48 per cent. had marked lowering of pressure, 37 per cent. a moderatd lowering, in 7 per cent. a tension above 130 mm. Hg. and in 8 per cent. the pressure was normal.

Of the febrile and afebrile cases the following table will give Schnitter's results:

Systolic pressure.	Febrile. Per cent.	Afebrile. Per cent.
60 to 100 mm. Hg.	65	22
100 to 115 mm. Hg.	30.5	48
115 to 130 mm. Hg.	1.5	19
Above 130 mm. Hg.	3	11

[1] Beitr. z. Klin. d. Tuberkulose, 1912, xxiii, p. 233.

In other words, the febrile cases showed lowered blood-pressure three times as often as the afebrile subjects. There is no constant relation between the pressure and the extent of fever, nor is there any ratio between the blood-pressure and pulse rate. The blood-pressure is usually higher toward evening. Loss or gain of weight does not seem to affect the pressure, nor does increase of blood-pressure go hand in hand with improvement, and hypotension with a change for the worse, although when a case is arrested the blood-pressure rises.

As regards hemorrhages, Naumann says that of the cases with pressure of 100 mm. Hg. or below, 36 per cent. had hemoptysis, while persons with higher tension had pulmonary hemorrhage in 51 per cent. of the cases, but this opinion is probably inaccurate. Acute miliary tuberculosis, especially when the meninges are diseased, is associated with rise in pressure.

Peters[1] points out that in his sanitarium, which is 6,000 feet above sea level, the blood-pressures are higher than at sea level or at lower elevations. He does not believe that blood-pressure has any causal relation to hemorrhage, for if this were the case, more cases of hemoptysis would have been met with and, as a matter of fact, but two were seen. The degrees of involvement and pressure are not related, although there is a rise when improvement sets in, which he believes is a reliable prognostic sign. Emerson puts the case even more strongly.[2]

Other authorities believe that the more pronounced and advanced the tuberculosis, the lower the blood-pressure, and *vice versa,* and he also holds that a low pressure will help to differentiate tuberculosis in dubious cases. Prognostically it has some significance. The imminence of hemoptysis must always be suspected when the blood-pressure rises, and he advises measures to combat the hypertension.

One must conclude that unless there is some associated condition like arteriosclerosis or nephritis, there is always hypotension. The author does not hold with these enthusiasts, particularly of the French School, that hypotension is an early sign of incipient tuberculosis, irrespective of the

[1] Archives Int. Med., 1908, ii, p. 42. [2] Ibid., 1911, vii, p. 441.

pulmonary and other clinical findings, although it no doubt is a significant sign and should make us heedful that we do not rush too rashly into the danger of superficial examinations and opinions. As to the cause of the hypotension. little is known, but it is probable that toxic action on the vasomotor centre, the vasomotor nerves, and the heart muscle all play a role. Not to be forgotten are the secondary effects of the infection, as anemia, loss of weight.

Apart from the hypotension in its broad aspect, there seems to be such divergency of opinion as to its prognostic significance and its value as a warning sign of hemoptysis, that it is difficult to take a definite stand.

In children, the blood-pressure is low as it is in adults, increasing within one or two hours when the patient is transferred to the open air. If kept constantly outdoors the pressure becomes normal, and the more advanced the case, the lower the pressure indoors, and the greater the rise when put in the open air.[1]

Lead Poisoning.—High blood-pressure is a common feature of lead poisoning and it has been supposed to be due to the arteriosclerosis arising in time from the intoxication. The "arteriosclerosis" explanation is hardly correct as it has been shown that the vessels in plumbism suffer changes in the media (hypertrophy) and that the intima is unaffected. The changes in the artery have been thought by some to be the effect of a direct action of the lead on the muscle of the vessel, giving rise to hypertension, and by others, to be the secondary manifestations of nervous irritation called forth by the lead.

Without taking up the history and development of our present knowledge on the subject[2] it may be stated that typical colic is always associated with high blood-pressure. Inasmuch as the vascular constriction must not of necessity be of maximum degree the blood-pressure rise is a variable one. This rise is a gradual one, reaching its acme with the explosion of the pain, although a sudden rise occurs in some cases. There is no parallelism between the height of the pressure and the degree of pain, for Pal quotes

[1] Hoobler, Amer. Jour. of Dis. of Children, 1912, iv, p. 311.
[2] See Pal, "Die Gefässkrisen."

cases showing attacks of pain with a pressure of 110 to 120 mm. Hg. These were, of course, not severe attacks but were nevertheless typical in their manifestations. After the attack, or rather, during convalescence from the intoxication, the pressure gradually falls below normal and again rises to the normal. Pal believes that these colic-like attacks are due to constriction of the abdominal vessels, and proves it by the effect of amyl nitrite which aborts an attack. Other effects of the vascular constriction are seen in slowing of the pulse and accentuation of the second aortic sound. To this must be added cerebral symptoms, headache, vertigo, amaurosis, hemianopsia, deafness, aphasia, transitory palsies, and even eclampsia. Tanqueul has shown that 56 cases of eclampsia occurred in 72 cases of lead poisoning. Pal mention angina pectoris among the rarer symptoms of plumbism, but says he has never seen a case.

The author has had several cases of plumbism lately, and among these have been instances of hypotension. It is true, however, that the majority of the cases do have increased blood-pressure. Irritation of the sympathetic nerves is held to be the prime cause of the vascular constriction.

Gout.—Gout is considered to be a hypertensive disease, although no specific work has been done on the subject. Haig believes that the high pressure is due to the obstruction of the capillaries by uric acid in a colloid form, thereby increasing peripheral resistance.

Alcoholism.—During the first days of a chronic alcoholic subject in the hospital (abstinence from liquor) there is a relative hypertension which gradually falls in from three to eight days to normal. The diastolic pressure is but little raised, but the pulse pressure is increased, due to the increase of the systolic pressure.[1]

Bichlorid of Mercury Poisoning.—In a case recently under my care, for two days the blood-pressure was 132–94 and 129–88 on the respective days. The patient had taken $52\frac{1}{2}$ grains of mercuric chlorid. A decapsulation operation was undertaken, but the patient died in a few hours.

[1] Raff, Deutsch. Arch. für klin. Med., 1913, cxii, p. 209.

III. MISCELLANEOUS.

Acute Pulmonary Edema.—The belief is that this obscure condition is accompanied by hypertension. Amblard has studied the blood-pressure in patients before, during, and after the attacks, in several cases. These figures are perhaps best studied in tabular form.

	Before, systolic.	Before, diastolic.	During, systolic.	During, diastolic.
1.	280 mm. Hg.	210 mm. Hg.	222 mm. Hg.	190 mm. Hg.
2.	270 mm. Hg.	190 mm. Hg.	200 mm. Hg.	160 mm. Hg.
3.	250 mm. Hg.	180 mm. Hg.	190 mm. Hg.	160 mm. Hg.
4.	240 mm. Hg.	160 mm. Hg.	155 mm. Hg.	130 mm. Hg.

During the attack, it is seen that the two pressures fall but the maximum pressure becomes relatively lower than the minimum.

After the attack, the systolic pressure gradually rises, the diastolic remaining essentially the same, but the former does not attain the height it reached before the crisis.

Amblard interprets these findings as pointing to a functional insufficiency of the left ventricle as a cause of the pulmonary edema, this inefficiency being brought about by a raising of the diastolic pressure. The latter, indicates increase in peripheral resistance, and in order that there may be the normal difference between systolic and diastolic pressure, the systolic rises to such a degree that the left ventricle becomes tired, the pulse more rapid, and acute pulmonary edema is produced with a subsequent fall in systolic pressure. Precautionary measures directed toward preventing an attack are described.

Pneumothorax.—In penetrating wounds of the thorax giving rise to hemopneumothorax, there is usually a slow, full pulse, similar to that seen in brain injuries, and called pressure pulse or Druckpuls (Sauerbruch). The cause of the slowing of the heart with its rise of pressure has been held[1] by Sauerbruch to be due to the accumulation of

[1] Sauerbruch, Mitteil. a. d. Grenzgeb., 1904.

carbon dioxide in the blood and to irritation of the vagus. What the cause of the latter is, is doubtful, whether reflex or mechanical (collapse of the lung), but Kakowski[1] believes that in addition to the direct stimulation of the vasomotor centre by carbon dioxid and to the strong muscular contraction, there is a third factor to be seen in reflex stimulation of the vasoconstrictor nerve through the skin and pleura. The latter point he was able to emphasize by observing no rise in pressure after cutting the vagi, and he believes that the vagi, in pneumothorax, behave like a sensory nerve. Sensory nerve fibers have been found by Ramström to exist in the parietal pleura,[2] but none has been found in the visceral pleura.

Walther[3] has lately made some experiments undertaken to elucidate, if possible, the cause of the blood-pressure rise. He was not able to confirm the findings of Sauerbruch and others, of the role of the vagus in its production, but believes it is solely the function of the vasomotor centre. The slowing of the pulse is due to irritation of the vagi. In studying the effect of increased pressure in a closed pneumothorax, there was not a rise in pressure with moderate pressure, but when a great tension is suddenly produced, there is a fall, then a rise, and then a drop. For details concerning the physiology of these changes in arterial pressure the reader is referred to Walther's original article. The study of blood-pressure in artificial pneumothorax should be of great importance.

Respiratory Diseases.—The role that dyspnea plays in the elevation of blood-pressure in certain respiratory diseases can not be wholly neglected. It is true that dyspnea appears only on exertion, and that the blood-pressure, even when the patient is at rest, is raised. On the other hand, subjective dyspnea is but one expression of a defective interchange of gas, and one can conceive of lesser grades than that required to produce subjective discomfort, notably imperfect interchange of gases between the tissues

[1] Pflüger's Archiv, 1910, p. 134.

[2] Mitt. a. d. Grenzgeb., 1906.

[3] Deutsch. Zeitsch. für Chirurgie, 1912, cxix, p. 253.

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and the blood without any visual manifestation of the same. Just how great a part this plays, one can but surmise, but it should not be entirely disregarded. Hensen, indeed, believes that the dyspnea is the underlying cause of hypertension in respiratory disease, it being directly responsible for the latter.

(*a*) **Asthma.**—In judging of this disease, one must have in mind bronchial asthma as distinguished from cardiac asthma, renal asthma or any of the paroxysmal attacks of a secondary or reflex nature. There has been found an elevation from 100 mm. Hg. to 140 during the attacks returning to normal during the periods of remission. These elevations may be due to nervous influences and to forced expiration, both of which would raise the blood-pressure in the absence of dyspnea.

(*b*) **Chronic Bronchitis and Emphysema.**—Janeway and others have found hypertension in these conditions, but ascribe it to a latent angiosclerosis, rather than to dyspnea or any factor inherent in the respiratory disease itself.

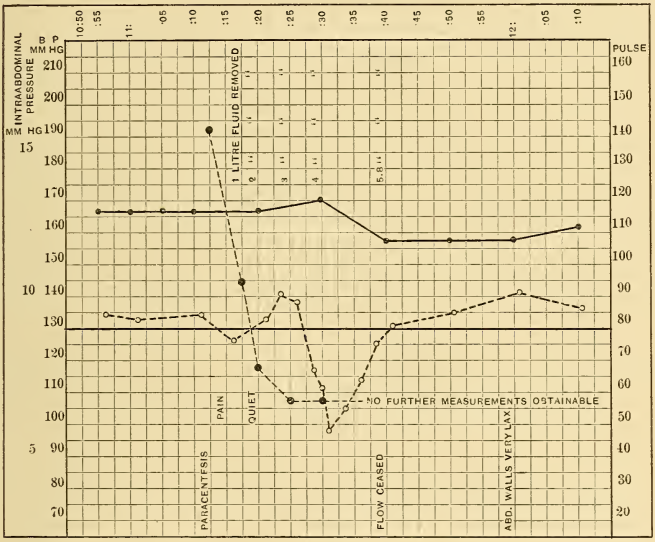
Pleural Effusion and Ascites.—Both of these tend to produce hypertension, but pleural effusions have a greater effect than peritoneal ecudations. After tapping, the pressure falls, there being more of a change with large pleural than with large abdominal effusion (see Fig. 34).

Raynaud's Disease.—Blood-pressure studies in cases of Raynaud's disease have as yet not been made in any great numbers. The writer has a case under his care, a laborer, aged thirty-six years, who has been suffering for six years with sudden whitening of his fingers. The blanching involves either one finger, or all fingers, and the attacks occur irregularly, sometimes every day and sometimes not for several days.

When I first saw the man the blood-pressure was 126–78 mm. Hg. During an attack the pressure was 154–86 mm. Hg. and just after the attack it fell to 118–72 mm. Hg. Before each attack there was a rise in blood-pressure, though this was not as marked as at the first observation. Between the attacks there was no constant blood-pressure figure, the blood-pressure varying between 118 and 146 mm. Hg.

(systolic). The diastolic pressure was little affected by the attacks.

FIG. 34



Abdominal paracentesis. (Cook and Briggs.)

Blood-pressure and Insurance.—Much valuable information has been afforded by the study of blood-pressure in insurance work. High blood-pressure is the only feature which has been given much attention, but it is gratifying to note that many of those making examinations are turning their attention to hypotension as well.

Inasmuch as pressures above 150 mm. Hg. generally spell nephritis, an insurance examiner should regard such pressures with suspicion. A well-known company has furnished statistics which show the wisdom of this statement, for of 365 cases rejected on account of high blood-pressure alone (170 mm. Hg.), 136, 35 per cent. later proved to be suffering with various serious ailments, any one of which would have provided a basis for rejection. The

mortality in these cases was 144.51 per cent., only a little below that of those cases rejected on account of high blood-pressure and one or more other impairment, in whom the mortality was 161.9 per cent.

Some interesting tables are furnished by Fisher.[1]

723 persons were rejected, with an average blood-pressure of 171.03 mm. Hg. (Table III). 358 of the 723 rejected cases, with an average blood-pressure of 171.73 mm. Hg., showed one or more other impairments than the high blood-pressure (Table IV). These impairments will be found in Table V. 365 cases, the remainder of the 723 rejected, showed an average blood-pressure of 170.36 mm. Hg. (Table VI). Of the 365 cases (Table VI), no other impairment was recorded on the application when received at the home office.

TABLE I.—Mortality of Accepted Risks with Systolic Blood-pressure of 140–149 mm. Hg., with an Average of 142.43 mm. Hg. All Ages.

Years.	Number.	Expected.	Actual.	Per cent.
1907	217	14.884	7	47.03
1908	652	41.221	21	50.94
1909	953	42.088	12	28.51
1910	846	25.215	18	71.39
Total	2,668	123.408	58	47.00

TABLE II.—Mortality of Accepted Risks with Systolic Blood-pressure of 150 mm. Hg. and Over; Average 152.58 mm. Hg. All Ages.

Years.	Number.	Expected.	Actual.	Per cent.
1907	87	6.194	6	96.87
1908	210	14.398	10	69.45
1909	163	8.432	3	35.57
1910	65	2.238	3	134.05
Total	525	31.262	22	70.37

[1] Medical Record, 1911, p. 818, and 1912, p. 1075.

Table VII shows the impairments which were discovered, or which subsequently developed in Table VI. Had it not been for the high blood-pressure, almost all of the cases would have been approved and the policies issued.

Table III.—Mortality of Applicants Rejected with Average Systolic Blood-pressure of 171.03 mm. Hg.
All Ages.

Years.	Number.	Expected.	Actual.	Per cent.
1907	40	3.193	10	313.18
1908	120	8.429	13	154.23
1909	210	11.095	16	144.21
1910	225	8.408	9	107.04
1911	128	2.163	3	138.69
Total	723	33.288	51	153.21

Table IV.—Mortality of Applicants Rejected with High Blood-pressure (average 171.73 mm. Hg.) and One or More Other Impairments.

Number.	Expected.	Actual.	Per cent.
358	16.680	27	161.99

Table V.—Impairments Recorded on Applications at Time of Medical Examinations of the 358 Cases Recorded in Table No. IV Above.

Arterio-sclerosis atheroma.	Heart murmur.	Heart hy-pertrophy.	Albumin and sugar.	Albumin in urine.	Sugar in urine.	Albumin and casts.	Casts in urine.	Nervous symptoms.	Prostatic disease.	Miscel-laneous.	Total.
53	69	17	10	111	15	10	6	21	5	41	358

Table VI.—Mortality of Applicants Rejected with High Blood-pressure Only. (Average 170.36 mm. Hg.)

Number.	Expected.	Actual.	Per cent.
365	16.608	24	144.51

Table VII.—Impairments Subsequently Discovered or Developed in the 365 Cases Recorded in Table No. VI Above.

Arterio-sclerosis, atheroma.	Heart murmur.	Heart hypertrophy.	Albumin and sugar.	Albumin in urine.	Sugar in urine.	Albumin and casts.	Casts in urine.	Nervous symptoms.	Miscellaneous.	Total.	Expected.	Actual.	Per cent.
7	10	4	3	20	5	36	16	6	16	123	5.867	8	136.35

The practising physician can well learn a lesson from his confrère in the insurance office, and forecasts concerning the future health of patients with hypertension should be tempered with marked conservatism.

No one can deny the seriousness of hypertension as far as insurance work is concerned. It would be a matter of no little importance if statistics were collected having hypotension as a basis. For instance, in view of the common association of hypotension with tuberculosis, it would be valuable to know what proportion of cases rejected on account of low blood-pressure and doubtful physical signs later developed tuberculosis, or what relation hypotension bears to resistance against disease. With the number of applicants and the routine estimation of blood-pressure it would not be difficult to collect statistics on hypotension which would be of the utmost value.

CHAPTER IX.

BLOOD-PRESSURE IN NERVOUS DISORDERS.

I. GENERAL CONSIDERATION OF INCREASED ARTERIAL TENSION IN DISEASES OF THE CENTRAL NERVOUS SYSTEM.

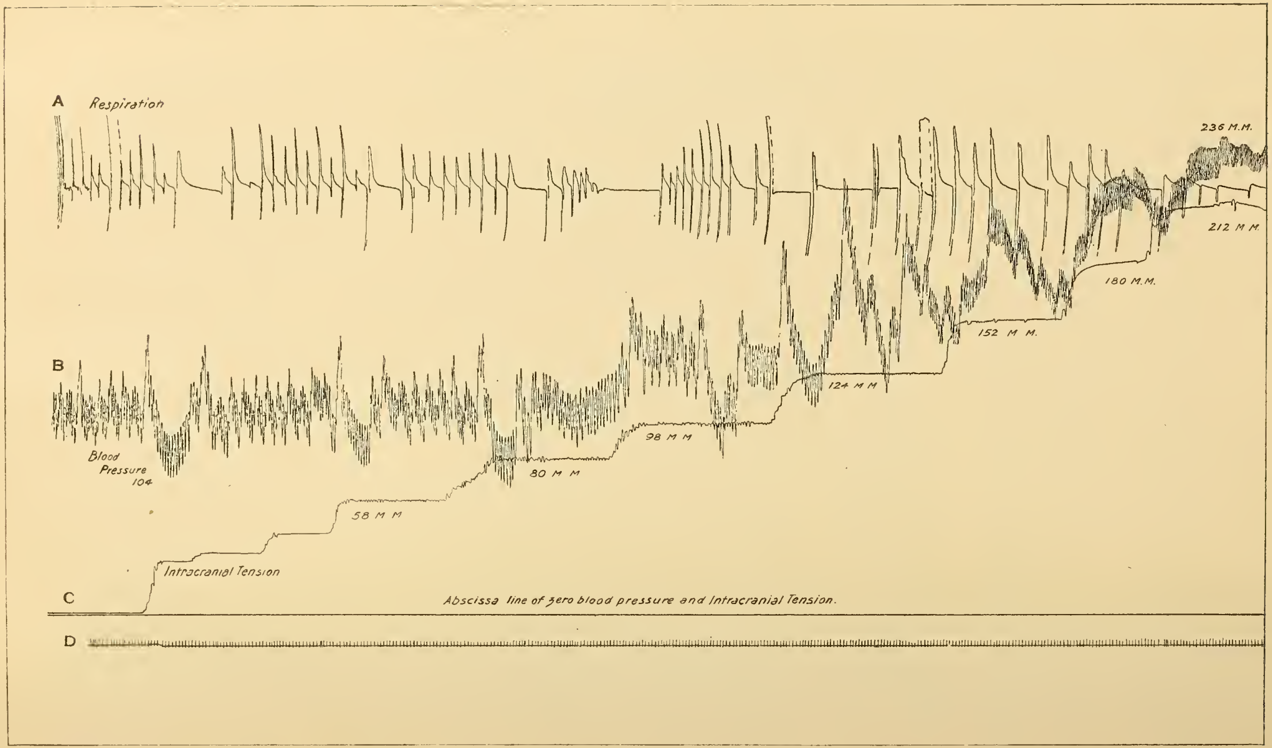
Many of the diseases of the brain may be divided into broad groups, the first comprising processes causing local compression of the brain; second, processes causing general compression of the central nervous system. The first group, then, would include new growth, abscess, blood clot, abnormal collection of fluid in one part of the cerebrospinal canal. In the second class of cases are found acute cerebral edema arising from traumatism, acute hydrocephalus, meningitis, subdural hemorrhage.

Experimentally the conditions of local and of general compression have been most thoroughly studied.[1] The effects of local compression vary a great deal according as the compressing agent in close proximity to or remote from the fourth ventricle, and these, from the stand-point of blood-pressure estimations, are of variable importance.

It is not until the intracranial tension approximates the blood-pressure that the pressure symptom-complex (Cushing) is produced (Fig. 35). It was formerly supposed that when such a degree of pressure was attained, death ensued, but has been shown quite definitely that this is not so, but that there occurs a stimulation of the vasomotor centre, which occasions a rise of blood-pressure to such a degree that the high intracranial tension is overcome. If the brain cortex be examined when intracranial tension and blood-

[1] Cushing, Amer. Jour. Med. Sci., 1902, cxxiv, p. 375; 1903, cxxv, p. 1017.

Fig. 35



Fragment of a chart of a "general compression" experiment on a dog. Showing a gradual increase in intracranial tension from zero to 212 mm. of mercury, the blood-pressure meanwhile increasing from its normal level at 104 to 231 mm. of mercury. Note continuance of respiration, absence of vagus effects, etc.

pressure begin to approximate, the rosy color becomes a grayish-yellow hue, and there seems to be little if any circulation between the arteries and the veins. As soon as the blood-pressure begins to rise, following stimulation of the vasomotor centres, the red color returns, the centres are again nourished, and respiration, which has ceased, recommences. If increase of intracranial pressure be carried beyond the new blood-pressure, the latter rises again, and by repeating this process the blood-pressure may be elevated two or more times its original value. Pressure can be carried to 250 mm. Hg. and maintained there for some time without any sign of vasomotor fatigue.

When the intracranial tension is suddenly brought to a height exceeding the blood-pressure, instead of gradually, as in the foregoing, the vagi are tremendously stimulated, causing cardiac arrest for a period exceeding thirty seconds at times. If this suddenly increased tension is kept up, the heart beats again and the blood-pressure gradually ascends to meet the level of intracranial tension. Cushing thinks this experiment imitates very closely the phenomena seen in clinical conditions. Just how long the reaction can be carried on varies a great deal, the more slowly the pressure is increased, the longer will the vasomotor centre respond, but after a time it weakens, there is dilatation of the vessels in the splanchnic area with resultant fall of blood-pressure below that of the brain, there is asphyxia of the respiratory centre and death ensues with a rapidly beating heart and a low blood-pressure. It has been thought that death in cases of high intracranial tension, respiration stops before the heart, but probably behind the respiratory failure there is vasomotor collapse, the vasomotor mechanism having become exhausted from maintaining the blood-pressure above the intracranial tension.

To summarize briefly, it may be stated that in a slowly forming intracranial condition blood-pressure is not elevated, whereas, in acute cerebral compression (extravasation of blood), there is always increase of arterial tension. In the latter case there is a definite regulating mechanism whose mission it is to maintain the blood-pressure above the intra-

cranial tension, and thus to prevent a bulbar anemia which is fatal (respiratory failure). A less important function of this vasomotor regulating mechanism is seen in the slowing of the pulse, although it does not play any role in the total blood-pressure reaction.

So much for the experimental side. In summing up his memorable monograph in Nothnagel's *Specielle Pathologie und Therapie,* 1901, lx, 3, p. 186, Kocher gives the following classification of the stages of compression seen in the clinic:

1. *Stage of Compensation.*—This corresponds to the stage of latent intracranial pressure, which formerly was so much talked about but which possesses no "latency" at all, as one may at this time find in man signs of pressure such as choked disk and in animals escape of liquor cerebri from the nose, and increase of venous pressure. The usual signs of this stage are insignificant, however, as the escape of cerebrospinal fluid and compression of the sinus and veins can compensate for the increased tension. There is practically no increase in blood-pressure.

2. *Beginning Stage of Manifest Pressure.*—If the cerebrospinal fluid has escaped from the skull and if the veins are as much compressed as they can be without absolute occlusion, then the second stage begins. The side channels of venous escape being blocked off there results venous stasis and a lessening of the blood-flow through the capillaries (dysdiämorrhysis). Signs of compression appear together with symptoms of irritation of the meninges: headache, vertigo, pain in the limbs, unrest, tinnitus aurium, altered sensorium with excitement, delirium, jacitation and sleep disturbed by dreams.

3. *Height of Manifest Pressure.*—The capillaries emptying themselves so slowly, there results cerebral anemia. If there is local compression, the anemia is localized, but lasting and local paralytic signs are observed such as monoplegia, hemiplegia, aphasia, hemianopsia, etc., according to the area compressed. There is a ready tendency of the anemia to invade other portions of the brain cortex, and the effects of these may be momentary and intermittent. Provided

the anemia does not encroach on the medulla, there is no immediate fatal outcome, but when the latter is invaded, death follows through bulbar anemia. The reason that death does not result earlier is on account of Cushing's blood-pressure reaction consisting of excitation of the vasomotor centre with rise in blood-pressure, strong enough to overcome the intracranial tension, and then to drive blood again through the capillaries. This anemia is intermittent, as a matter of course, and there is always a balancing with adiämorrhysis and endiämorrhysis on either end. Respiration is altered, so that in the stage of anemia of the centre, it stops, and in the stage of blood flow, it begins. During normal circulation the vasomotor centre is relatively quiet but is put into activity immediately when there is anemia. The vagus is stimulated to a great degree by the dysdiämorrhysis.

4. *Stage of Paralysis.*—If there follows still more pressure in the vessels, the blood-pressure is able to overcome it only in systole, while in diastole the capillaries remain empty. Now the change which was rung between adiämorrhysis and endiämorrhysis in the third stage is played between adiämorrhysis and dysdiämorrhysis and symptoms of general cerebral involvement follow: tremor, nystagmus, anisocoria, arrhythmia, irregular respiration, and finally dilated pupils, intermittent, snoring respiration and rapid, running pulse with low blood-pressure.

The reader is referred to a series of cases reported by Cushing[1] which exemplifies most beautifully Kocher's classification. Cushing emphasizes the advantage of blood-pressure estimation in case of intracranial injury.

A point which Cushing presses and one to which most clinicians will most heartily subscribe, concerns treatment of cases of intracranial hemorrhage with a high, bounding pulse. His words are indeed worthy of literal quotation. "The therapeutic measure generally advocated in cases of intracranial hemorrhage with high, bounding pulse is a purely symptomatic one, namely, to 'bleed' the patient

[1] Amer. Jour. Med. Sci., 1903, cxxv, p. 1017.

in order to lower blood-pressure, the idea being, that the persistent high tension is the cause rather than the result of the hemorrhage. If the interpretation of this experimental work is not at fault, such an abstraction of blood with the idea of lowering arterial tension would be absolutely contra-indicated, since the high blood-pressure is only an indication of Nature's effort to overcome the degree of intracranial pressure brought about by the foreign body in order to ward off an anemic condition of the bulbar centres. Of course the tension of the foreign body in this case, communicating as it does with a ruptured artery, is equal to that of the arterial tension, and were it not for its remoteness ordinarily, from the medulla, death would almost immediately ensue, just as it does when a hemorrhage takes place in the neighborhood of the medulla or reaches the fourth ventricle though ruptured into one of the lateral ventricular cavities.

"In the course of many of the experiments on compression the effects of blood-letting (the skull still being intact) were tried during the various stages of compression. At no time could any beneficial influences be seen, and if blood was withdrawn from the general circulation at a time when there had been a marked rise in general arterial tension to overcome cerebral anemia, and in amounts sufficient to lower this arterial tension, the results were almost always diastrous unless a certain amount of reserve power remained in the vasomotor centre which could make return the arterial tension to its former level. I would not mean to deny that symptomatic improvement ever follows blood-letting in cases of apoplexy in which the symptoms have not called forth pronounced symptoms referable to the vital bulbar centres, but when such is the case, anything which tends to lower arterial tension without an associated opening in the skull to correspondingly lower intracranial tension, is hazardous to say the least."

II. EFFECT ON BLOOD-PRESSURE OF LUMBAR PUNCTURE.

In many pathological states of the central nervous system, especially of the brain, tumors, cerebral hemorrhagic meningitis, cerebral and cerebrospinal meningitis, both serous and purulent, there is in addition to the increased pressure of the cerebrospinal fluid, increased arterial pressure. If the cerebrospinal fluid is allowed to escape until its pressure reaches normal, the arterial pressure will fall likewise to normal or below normal, depending on the amount of liquid withdrawn. The pulse, which has been 40 to 50, becomes more rapid after the puncture, reaching normal or slightly exceeding it.

The arterial pressure remains lowered after lumbar puncture, the length of time depending on just how rapidly the cerebrospinal fluid reaccumulates. This is particularly well seen in cases of cerebrospinal meningitis, where it may be shown in the same individual that arterial pressure rises every time the cerebrospinal fluid accumulates to such a degree that its pressure is raised. When the disease progresses favorably and the cerebrospinal pressure diminishes, arterial pressure becomes again normal.[1]

Lafite-Dupont and Maupetit[2] found that raising a pressure in the labyrinth raised the arterial pressure, and when the pressure was raised in the cerebrospinal canal, it was again raised. Removal of fluid lessened pressure. In man lumbar puncture can cause a drop of arterial pressure from 40 to 80 mm. Hg., this drop lasting several days.

The question of blood-pressure in lumbar puncture has been most carefully studied. The effect of pain, mental disturbances and muscular contractions have been noted, and these are of themselves interesting, as showing that the skin puncture is followed in every case by a rise in pressure which varies with the amount of pain resulting from the

[1] Parisot, Compt. Rend. Soc. de Biol., 1909, p. 939; cf. section on Epidemic Cerebrospinal Meningitis.

[2] Compt. Rend. Soc. de Biol., 1905, p. 677.

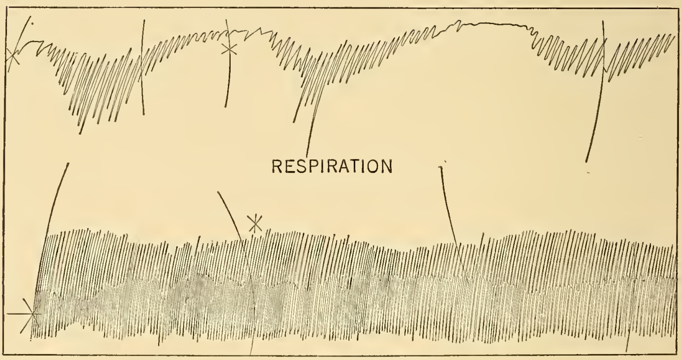
manœuvre, and with the degree of consciousness of the patient. A greater and a more marked rise is associated with the puncture of the dura, which rise is just as marked if the patient is unconscious as when he is fully alive to his surroundings. Puncture in the dura in a completely anesthetized dog exhibits the same phenomena, thus disproving any view that the alteration is due to pain or disturbance of the higher centres. The effect is probably vasomotor and indicates a definite influence on the medullary centres.

Withdrawal of the spinal fluid is followed by an immediate fall of pressure, but there is an almost immediate rise so that for at least twenty minutes after the operation, the pressure is higher than normal.

III. CHEYNE-STOKES RESPIRATION AND BLOOD-PRESSURE.

Pollock[1] has confirmed the findings of Cushing and of Eyster in 15 cases of Cheyne-Stokes respiration arising

Fig. 36



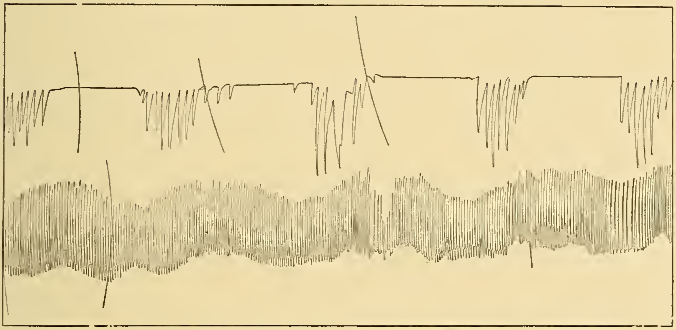
High blood-pressure during apnea; infarct of lung.

from various causes, including cerebral hemorrhage, cerebral and general arteriosclerosis, myocarditis, decompensated

[1] Arch. Int. Med., 1912, ix, p. 406.

heart lesions, pneumonia, infarct of the lungs, and nephritis. Two curves are reproduced from his article, showing the high blood-pressure during apnea in infarct of the lung (Fig. 36), and low pressure, during apnea in a case of cerebral hemorrhage (Fig. 37).

FIG. 37



Low pressure during apnea; cerebral hemorrhage.

It may be definitely stated that in Cheyne-Stokes respiration due to increased intracranial tension, the blood-pressure begins to rise slightly before respiration commences, becoming highest at the height of rapid breathing, and then falling to reach its lowest point during apnea. In cases in which there is no increased intracranial tension, the pressure begins to fall when respiration commences, rising and becoming highest during apnea.

IV. INSANITY AND OTHER CONDITIONS.

Melancholia.[1]—In cases of acute melancholia the average pressure is 150 mm. Hg., the range being between 140 and 160. Usually the more profound the depression, the higher

[1] Craig, Lancet, 1898, i, p. 1742.

the pressure, and the latter seems to be higher in women than in men. A curious coincidence was the mental improvement as the day went on, associated with lowering of blood-pressure. Craig publishes two interesting tables, the one illustrating the former observation, and the second showing blood-pressure before and after treatment; improvement and even recovery being noted in every case in which there was a fall of blood-pressure. It may be stated that there was lowered blood-pressure in every case which improved or recovered. Others insist that the pressure is *always* elevated in melancholia, but Craig is not so emphatic, as in depression with motor restlessness, the so-called agitated melancholia, the blood-pressure readings are somewhat varied, although when restlessness becomes extreme the blood-pressure is sometimes low.

Alexander[1] divides melancholia into four varieties; simple melancholia, acute passive melancholia, acute demonstrative melancholia, and chronic melancholia. There is marked elevation of the blood-pressure in the simple form of the malady but in the acute passive type it is always high (155 mm. Hg., Hill and Barnard). The course of the blood-pressure is irregular in agitated melancholia, while in chronic melancholia the blood-pressure is generally low, an elevation occurring when the mental symptoms became more acute.

Regarding the role blood-pressure plays in insanity, Craig favors the view that the high pressure is the cause of the mental depression, and Alexander, that the high blood-pressure is due to toxemia arising from the retention of waste products of tissue metabolism. The view of Craig does not make a strong appeal, as mental depression occurs with hypotension and many cases of hypertension are seen with no sign of melancholia. Surely hypertension alone cannot explain the mental state.

Acute Mania.—A constant lowered tension seems to be the rule in acute mania, the average being 105 to 110 mm. Hg. (Craig). Pilcz has also found lowered pressure, but

[1] Lancet, 1902, ii, p. 18.

gives the very good advice, to bear in mind the possible association of a chronic nephritis, which will upset all calculations. Alexander studied 16 cases of acute mania, in which the readings obtained were invariably above normal. Alexander noted improvement with fall of pressure and Craig with rise to normal. Baths of seven and eight hours' duration seemed to have had in Craig's hands a most soothing effect on the patient and also caused the pressure to rise from 105 to 120 and 130. Before the bath the patient was very excited and after the bath she was quiet and reasonable. Such divergent views prevent one from having definite ideas on the subject.

General Paresis.—The arterial pressure (Gärtner) is at the lowest normal figures in this disease but as the disease progresses, it falls very low (50 mm. Hg., Pilcz). With remissions of good health, the pressure rises. A sudden fall is common just before death and Pilcz claims in this sign a very serious and reliable omen of impending dissolution. Nephritis of course, vitiates these findings. Craig has had limited experience with unsatisfactory material, but believes the pressure is high at first, but later, is low and suggests that cholin arising from the disintegrating nerve tissue may be the cause of this depression of tension.

In connection with the subject of general paresis it might be added that in treating this condition by means of salvarsan, McKinniss[1] found no rise in pressure even when 195 c.c. saline salvarsan solution was injected.

Circular Insanity (Folie circulaire).—Judging from observation on other types of mental disease it seems more than probable that the blood-pressure will vary with the mental condition; that is, that it will be raised when the person is depressed, and lowered when the subject is excited, but that during the period of health it will remain normal.

In cases of stupor, the reading is higher than in melancholia, but when the stupor is secondary to mania the blood-pressure is low, owing to the exhaustion.

[1] Medical Record, 1912, lxxxii, p. 100.

It has been pointed out that suicides are common about 6 A.M., while toward evening the misery is less or has disappeared and the patient employs or amuses himself, and it is hinted that these mental states may follow the physiological decline of pressure which takes place toward evening. On the other hand, the maniac is at his best in the morning, and the excitement becomes worse about 4 P.M.

Myasthenia Gravis.—The pressure was found to be what Steinert[1] calls the lowermost limit of normal (100 to 120 mm. Hg. R. R.).

Neurasthenia.—There being divergent views regarding the type of case one should place under the diagnosis of neurasthenia, it is difficult to interpret the blood-pressure readings with any degree of satisfaction. The more one reads of neurasthenia and the more so-called instances of this condition one sees, the less certainty is felt in the correctness of this diagnosis.

Stursberg[2] and Stursberg and Schmidt[3] have studied the effect of exercise on blood-pressure in neurasthenic states. In 1907, Stursberg wrote that neurasthenic individuals showed the same qualitative differences in blood-pressure after exercise that normal individuals exhibit, but that there was an increase in the dynamics of the heart. During rest, the blood-pressure and pulse-pressure were higher than in healthy subjects. In their later paper, Stursberg and Schmidt arrive at practically the same conclusion.

Their statement that blood-pressure is raised in neurasthenic patients, is open to some criticism, as the medical profession, especially neurologists, are far from being of one opinion regarding the diagnosis of neurasthenia.

Epilepsy.—During the time of an epileptic seizure, the pressure rises to a great height, and remains high during the paroxysm, falling below normal when the attack has subsided. This postepileptic depression remains for many hours (eight, ten, twenty-four) following a convulsion.

[1] Deutsch. Arch. für klin. Med., 1903, lxxviii, p. 346.
[2] Archiv für klin. Med., 1907, xc, p. 548.
[3] Münch. med. Woch., 1913, p. 174.

When the seizures are rapidly repeated, the arterial tension thereafter remains low for days. The explanation for the great rise in pressure is the same as for violent muscular exertion.

The differential diagnosis between epilepsy and eclampsia by means of the sphygmomanometer will be described on page 168. The same general rule guides us in distinguishing between epilepsy and uremia, namely, that in the former the blood-pressure returns to normal after the attack, while in the latter it remains high throughout the convulsion and the coma.

Tabes Dorsalis.—As far as blood-pressure determinations in disease of the spinal cord are concerned, observations seem to be limited to tabes dorsalis.

It is the belief that the crises of tabes are associated with increase of blood-pressure, but that this is not the invariable rule, is shown by Pal[1] in two cases. The first had a drop in pressure from 125 mm. (Gärtner) to 80 mm., and the second, from 155 to 110. Pal believes that the posterior horns of the spinal cord have vasodilator fibres (Strickler), which are irritated in the tabetic process. It is true that since tabes is entirely a disease of the posterior roots and if irritation caused vasodilatation, low blood-pressure should be the rule, which it is not, by any means. This is explained by supposing vasodilator fibres and vasoconstrictor fibres, and, depending on which is the nerve irritated, there is a fall or rise of blood-pressure.

The subject of blood-pressure in tabes is fully described in Pal's book, "Gefässkrisen," 1905, in which work an attempt is made to explain the critical pain on sudden arterial convulsion. The blood-pressure rises *pari passu* with the dimension of the crisis, provided there is no associated cardiac weakness. The amount of pressure is variable but it may rise 50 per cent. of the original tension, attaining often a percentage of 150 and even more. In one case reported the blood-pressure rose from 90 mm. to 200 mm.

Although tabetics are commonly afflicted with arterio-

[1] Wien. med. Woch., 1904, p. 2.

sclerosis, the blood-pressure in the interim between crises, is not raised. Just before the crisis a rise in pressure may be observed, and the crisis is discharged coincidently with a high pressure. Therapeutically this has been substantiated by giving amyl nitrite, which induces an immediate cessation of pain. Sodium nitrite had the same effect in milder cases. With the onset of menstruation, especially when the hemorrhage is severe, the crises are inhibited. All these facts, and many more, Pal uses to develop his theory that the lancinating pains are produced by local arterial spasm.

CHAPTER X.

BLOOD-PRESSURE IN OBSTETRICS.

Pregnancy.—Throughout pregnancy blood-pressure remains normal. When labor is instituted, the pressure rises, and this rise in pressure is in the form of waves corresponding to the periods of uterine contraction. In the first labor pains the pressure may rise to 210 to 220 mm. Hg.,[1] returning to normal during the remissions. Later in the labor, pressures of 230 to 240 are seen, but in the last stages of labor there is but little fall of pressure, the greatest being drops of 20 to 30 mm., never returning to normal. When the child is born, pressure falls suddenly and completely, and remains normal from there on. When labor has been particularly difficult, with considerable hemorrhage, the pressure after delivery may be lower than normal but this rises, so that on the following day it is again at its normal level. Vaquez has never observed the *progressive* return of which some authors write. He emphasizes most strenuously, that there is not, either antepartum or postpartum, a physiological hypertension, and that only during labor itself, is the pressure elevated as the necessary result of effort, and he insists that any hypertension before or after labor is an index of a pathological state.

Fig. 38 shows the effect on blood-pressure of some of the stages of pregnancy.[2]

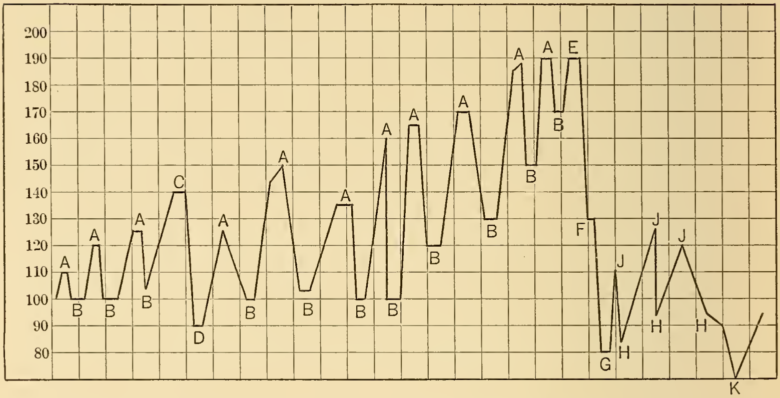
With the first pains there is a difference of 10 mm., the highest blood-pressure occurring simultaneously with the maximum of the contraction. When the latter becomes stronger the differences are greater, but there is a return to

[1] Potain and Gärtner's instrument, Vaquez. Bull. de la Soc. d'Obstet. de Paris, 1906, ix, p. 30.

[2] Monatsch. für Geburtshilfe und Gynekologie, 1901, xiv, p. 370.

normal in the remissions. Immediately after the rupture of the membranes there is a drop in pressure 10 mm. below the original, more when there is hydramnios. When the pain becomes more frequent, and relaxation of the uterus is not complete the pressure does not fall to normal between the attacks, but rises from pain to pain. Whereas the difference in pressure has been 90 mm., when the time for expulsion of the infant is at hand it is but 20 mm. The highest blood-

Fig. 38



Schematic representation of blood-pressure during labor: *A*, pain; *B*, pause; *C*, rupture of membranes; *D*, immediately thereafter; *E*, severest pain, appearance of head; *F*, escape of head; *G*, immediately after the birth; *H*, uterus firm; *J*, uterus soft; *K*, immediately after delivery of placenta.

pressure is attained at the birth of the head, usually 60 to 100 mm. above the original pressure. After the birth of the child, pressure falls again, 10 to 20 mm. below the original level, rises again for one-half minute, and then falls again. After this there are variations observed, corresponding to the uterine contractions; the stronger the contractions, the lower the pressure, the differences being 5 to 20 mm. Hg. Hemorrhage has little effect on the pressure, but delivery of the placenta causes a fall below the original level.

Eclampsia.—The first important work on this subject was done by Vaquez and Nobécourt,[1] and their observations have not been seriously refuted. They showed at that time that hypertension is a premonitory sign of eclampsia. Albuminuria seemed to cause no serious concern so long as the pressure remained at the normal level, 130 to 140 (Potain), and they held that the prognostic sign of the greatest importance is the hypertension and not the urinary findings. Vaquez[2] has again entered the field, and his results today are the same as those of nine years ago, namely, every woman, who, in the course of pregnancy or after delivery, exhibits increased arterial tension, is in danger of eclampsia, whether the urine contains albumin or not. During eclampsia, persistence of hypertension indicates return of a crisis even if other signs are reassuring. Even when eclampsia appears to be well controlled cure can only be stated as having been accomplished when the arterial pressure returns to normal.

High blood-pressure above 150 mm. is always a warning sign, and one should be on his guard against eclampsia.[3]

In a study of 145 pregnant women, repeated blood-pressure estimations (1136 readings) disclosed a variable blood-pressure in health between 90 and 132, the average being 118 mm. Hg. (Stanton—wide cuff) (Bailey). Bailey[4] agrees with Janeway, that a blood-pressure of 160 mm. Hg., does not exist in normal pregnancy. I believe 160 mm. Hg. is too high a limit, and prefer to view with suspicion any blood-pressure that persistently exceeds 140 mm. Hg., by the auscultatory method. This statement must not be misinterpreted. Sudden rises in blood-pressure from excitement, exertion, change of posture, digestion and the variations which we know occur in the pregnant as well as in the normal woman, are of course excluded, as they do not give rise to persistent hypertension. Vaquez's warning should sound in every ear, that "every woman,

[1] Soc. Méd. des hôp., January 9, 1897.
[2] Bull. de la Soc. d'Obstet. de Paris, 1906, ix, p. 34.
[3] Haussling, Jour. Med. Soc. of New Jersey, 1912, p. 242.
[4] Bailey, Surg., Gyn., and Obst., 1911, xiii, p. 505.

who in the course of pregnancy, or after delivery, exhibits increased arterial tension, is in danger of eclampsia." Bailey remarks, "while it is generally known that cases of eclampsia, at the time of the convulsion usually have high blood-pressure, still convulsions occur when the pressure is as low as 155." This 155 mm. Hg. is evidently too high a limit and a safer standard would appear to be 140 mm. Hg., although Bailey gives 150 mm. Hg. The latter made his readings by the palpatory method, which would make the pressure about 160 mm., when estimating by the auscultatory method.

Eclampsia and Uremia.—The close resemblance between the symptomatology of eclampsia and that of uremia has led to the erroneous belief that eclampsia is always a condition secondary to renal change. This is incorrect, as postmortem records of cases show. One will find true eclampsia cases with albuminuria, but with no demonstrable renal change, and others where there was no antemortem or postmortem evidence of nephritis. Chirié[1] reports a puzzling case of eclampsia who died finally of uremia, but in which the two conditions were definitely distinguishable.

Eclampsia and Epilepsy.—Chiriè[2] also reports a case where there was a nice point in diagnosis between eclampsia and epilepsy. The patient was a girl, aged seventeen and a half years, a primipara, in labor, who had had five convulsions. After admission, four more crises were noted. The differential diagnosis was based on answers obtained from the mother as to the girl's childhood, but especially on the blood-pressure, which remained normal, before and after the birth of the baby. In epilepsy, the pressure is high during the attack, but falls thereafter to normal or subnormal, whereas in eclampsia, hypertension is a constant feature.

Lactation.—After the birth of the child the blood-pressure generally falls to about 110 mm., but rises slowly and reaches its height the third or fourth day after delivery, when the milk appears. The pressure then is about 125 to 130

[1] Bull. de la Soc. d'Obstet. de Paris, 1907, x, p. 86.

[2] Ibid., p. 82.

mm. Hg. When lactation is established, the pressure falls again and approximates that observed before the milk was secreted. It will be thus seen that although a persistent postpartum hypertension is a sign of disease, yet, nevertheless, there is a temporary rise of blood-pressure which is purely physiological and which returns to the antepartum level when milk is seen. It seems preferable to speak of this postpartum hypertension as a relative or functional hypertension.

CHAPTER XI.

BLOOD-PRESSURE IN SURGERY.

Importance of Blood-pressure Records in Surgery.—The wide use of the sphygmomanometer among surgeons is a sufficient testimonial of its value during operative procedures. As a means of estimating shock there is no means equivalent to the blood-pressure instrument and all investigators are agreed that a drop in pressure is the best index to the beginning of the dreaded surgical complication. During every operation a chart should be kept on which should be recorded, every five or ten minutes, the pulse and the respiration, and every ten minutes the blood-pressure. Bloodgood[1] has described the details of a method whereby the coöperation of the surgeon, anesthetist and the chart keeper can best be obtained.

Experimental studies and practical results have shown that changes in blood-pressure are a better index to the extent of the trauma of the operation than are the pulse and respiration. Blood-pressure gives warning of shock before changes are observed in the pulse and respiration, and by heeding this warning, proper treatment may be instituted in time.

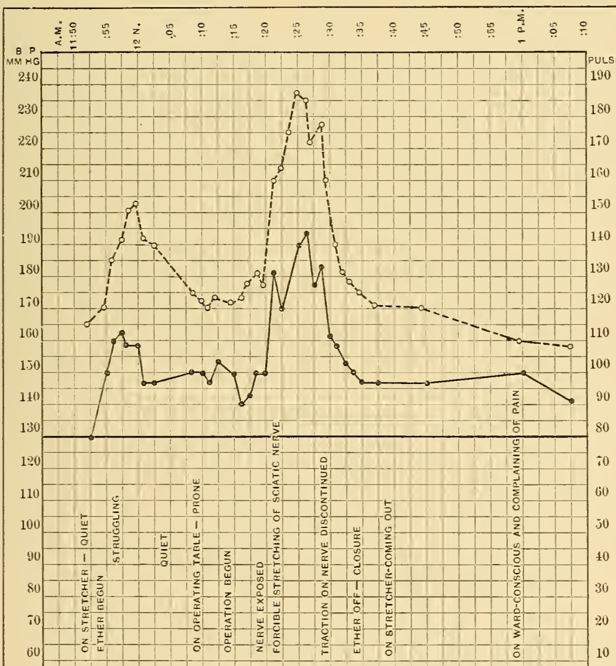
Effect of Anesthetics on Blood-pressure.—Ether.—A committee appointed by the Royal Medical and Surgical Society, in 1864, to investigate the effect of ether on blood-pressure reached the conclusion that ether was without any noteworthy effect on blood-pressure. Since then quite an opposite opinion has been obtained. Kemp[2] found in animals that ether raised blood-pressure, even when anesthesia is but slight, and that when deep anesthesia is ob-

[1] Progressive Medicine, December, 1912, p. 221.

[2] New York Med. Jour., November, 1899.

tained, there is again a rise. Cook and Briggs report a rise which they believe is due to the irritating effects of the vapor on the mucous membrane of the air passages. A second rise is seen in the second stage, probably due to muscular effort and the general excitement. They experi-

FIG. 39



Forcible stretching of left sciatic nerve for sciatica. (Cook and Briggs.)

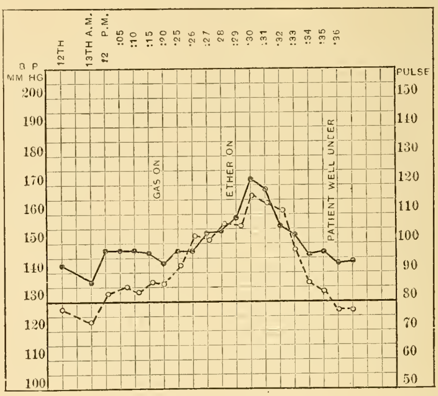
mented on themselves and found that when there was no general excitement, there was but little change in blood-pressure. When anesthesia is deep there is a slight fall which is more pronounced if there is sweating, but never very low unless there is some degree of shock; when the

subject begins to "come out" of ether there is generally a rise in pressure. The initial rise in blood-pressure is well shown in Fig. 39 reproduced from Cook and Briggs' article.

Chloroform.—All observers are agreed that there is a fall in pressure with chloroform, this being due to an effect upon the heart and not to an effect upon the vasomotor centre. Chloroform stimulates rather than depresses this centre, for injection of the drug into the cerebral arteries causes a rise in pressure which is still present when respiration ceases.

Cook and Briggs find that the action of chloroform is depressed from the start except in labor, which fact they explain as being due to the high blood-pressure occurring during labor.

Fig. 40



Anesthesia with nitrous oxid and ether. (Cook and Briggs.)

Nitrous Oxid.—There is a rise in pressure with nitrous oxid which is directly comparable to the changes seen in asphyxia (Fig. 40). There is no cardiac depression, however, until respiration has become very much interfered with. When the respiratory embarrassment takes place there is a fall in pressure which is due to impeded pulmonary circulation. When used preparatory to ether, there is a rise in pressure similar to that seen when ether alone is used, but the later secondary rise in tension is almost completely eliminated,

the patient passing from primary to deep anesthesia very quickly, thereby saving five to ten minutes of exhaustive struggling which would give rise to hypertension.

Since the introduction of the use of light nitrous oxid anesthesia (analgesia) into dentistry, it might be well to study its effect on blood-pressure, and this the writer has done in a very few instances, but without significant result.

A. C. E. Mixture.—The effects of this mixture are very similar to those of chloroform.

Cocain.—In abdominal operations performed under cocain there is not so much alteration in the blood-pressure as is seen in operations under general anesthesia. Momentary pain or fear causes an irregular rise of blood-pressure. Cushing claims that spinal injections of cocain may induce hypotension through paralysis of the nerve fibres governing the splanchnic circulation.

Spinal Anesthesia.—Cases of high spinal anesthesia exhibit, before the operation, very high blood-pressures, due to mental anxiety. The lumbar puncture itself (see Lumbar Puncture), causes a rise, then a "preliminary fall," followed by the "main fall" which is more marked (paralysis of the thorax), and this in turn by a rise to the original pressure, as the paralysis subsides. The "preliminary fall" is due to three causes, flaccid paralysis of the abdominal and skeletal muscles; subsidence of the disturbance, caused by the lumbar puncture; and onset of mental calm. In "low anesthesia" the "main fall" is missing but the "preliminary fall" persists.[1]

Nitrous Oxid and Oxygen.—The method of Crile with nitrous oxid and oxygen is held by surgeons to be by far the safest method yet devised, especially when combined with infiltrations of novocain. When anesthesia is begun the blood-pressure rises from 5 to 15 mm. Hg. and even higher, if there is any struggling or cyanosis. The more quiet the patient and the less the cyanosis, the less the blood-pressure rises. After the first fifteen to twenty minutes,

[1] Gray and Parsons, Quart. Jour. Med., 1912, v, p. 339.

if the patient is quiet, the anesthetic well taken, and there is no cyanosis, the pressure falls slightly, 5 to 15 mm. Hg. (Bloodgood).

Blood-pressure During Surgical Operations.—It seems to make little difference as far as the effects of cutting on blood-pressure are concerned, whether the patient is anesthetized or not. All incisions irritate the nerve fibers and cause reflex vasoconstriction. If pushed beyond a certain point operative procedures may have the same effect as if the patient were not anesthetized; that is, there will be a fall in blood-pressure. This fall in pressure (shock) may be transient or permanent, but in either case it is a most serious sign, the transient hypotension marking the inception for shock and the permanent hypotension indicating that shock is well established. Of this we shall speak more fully when shock is discussed.

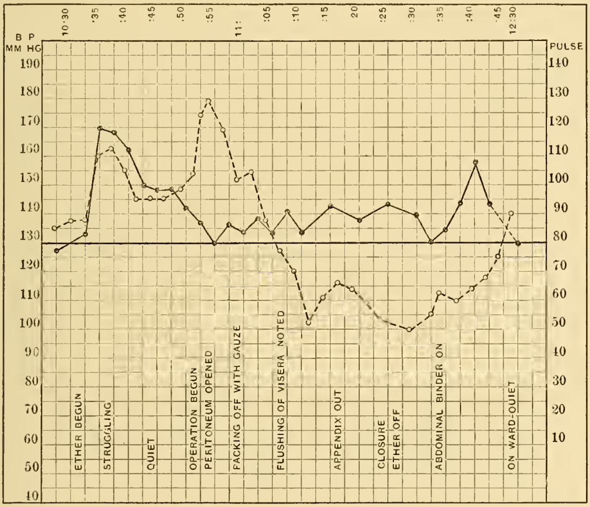
Operations on the Head and Neck.—There is no change in blood-pressure until the dura is reached, when irritation of this membrane causes a fall with considerable regularity. Elevation of the temporosphenoidal lobe produced a marked rise in the blood-pressure (Crile). Hemorrhage occuring from the meningeal artery, which was controlled by packing, caused a fall, and the latter was probably the combined effect of hemorrhage, irritation of the dura, water and the pressure of the brain. The amount of shock seems to be proportional to the amount of handling the brain receives and also the amount of hemorrhage and the duration of the operation.

In operations on the neck there is no distinct relation between the kind of operation and the condition of blood-pressure, provided there is no asphyxia, in which case, there is a rise in pressure. When the vagus had to be divided there was no effect on the blood-pressure provided the nerve was cut quickly (Crile).

Operation on the Thorax.—Excision of the breast for carcinoma in middle-aged subjects was attended by only moderate changes. In extensive operations on the axilla there was an irregular decline. The opening of an empyema with discharge of pus caused rapid fall in blood-pressure (Crile).

Abdominal Operations.—The chart reproduced from Cook and Briggs shows the effect of opening the abdominal cavity, there being a rise in pressure, when the peritoneal cavity was opened, above the initial rise of blood-pressure and above the rise produced by incising the skin.

Fig. 41



Laparotomy (appendectomy). (Cook and Briggs.)

The amount of handling and manipulation which the viscera receive directs the blood-pressure curve, there being a fall when much traumatism is inflicted. Packing the abdominal cavity with gauze or flushing out the cavity causes a decided decline. In ordinary simple appendectomy, but little change is seen, but severer operations such as excision of gangrenous bowel in strangulated hernia produced lowered pressure.

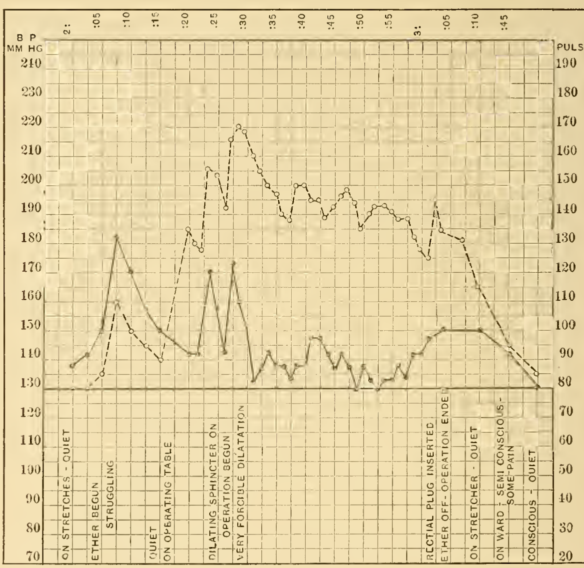
Genito-urinary Operations.—During operations for resection of the ovaries, the removal of ovarian tumors, excision of the tube for pyosalpinx, myomectomy, hysterectomy, both abdominal and vaginal, dilating and curet-

ting the uterus, and plastic operations on the vagina and perineum, in almost every instance there was a rise in blood-pressure (Crile). Practically similar phenomena were seen in the different peritoneal and vaginal operations. Crile made observations also during nephrectomy, nephrotomy, nephrorrhaphy and suturing of the ureter. Nephrectomy was followed by a rapid fall in pressure, while in simple nephrotomy but slight effects were seen.

In operations on the male genitalia, a fall in pressure was generally seen.

The following chart from Cook and Briggs shows the effect on pressure of the Whitehead operation for hemorrhoids.

FIG. 42



Whitehead operation for hemorrhoid. (Cook and Briggs.)

Operations on the Spinal Column.—Crile performed two laminectomies under cocain, and noted no change in pressure until the dura was reached, when a marked fall in pressure was seen.

Joachimsthal[1] has studied the effects of suspension on blood-pressure in orthopedic conditions and in cardiac lesion. Using the Dudgeon apparatus he made tracings in eight cases of vertebral deformity, scoliosis and spondylitis, and in cases of anemia and cardiac failure, and although his technique is not above suspicion he believes, that co-existing cardiac lesions, which have been heretofore regarded as a contra-indication are not affected by such treatment.

Cowl and Joachimsthal[2] subsequently made experiments with frogs and rabbits, studying the effects on blood-pressure of applying weights to the suspended animal, and believe that extension of the spinal column does not affect the blood-pressure to any noticeable degree until the weight applied is about equal to the animal's body weight, when a slight rise in pressure will be observed.

Operations on the Extremities.—Both Crile and Cook and Briggs have found a rise in pressure on stretching the sciatic nerve, which effect is well shown in the latter's chart. (See Fig. 38.)

Amputation of limbs following railway accidents was followed by marked fall, but in an amputation of a shoulder-joint, in which the trunks of the tracheal plexus were cocainized, thereby "blocking the afferent impulses, no material changes in the blood-pressure was noted" (Crile). Bone operations produced small variations, the greatest changes being seen when the periosteum was attacked.

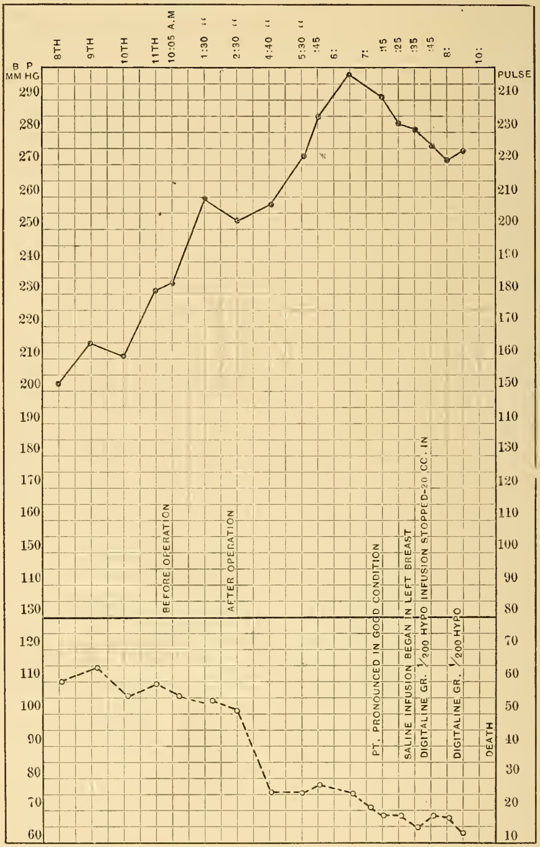
Blood-pressure in Surgical Complications.—(*A*) **Shock.**—As old as this subject is, and as much discussed as it has been, physiologists and surgeons are by no means in accord as to its cause. At the present time there are two leaders in the shock research, Crile and Henderson, and as both have their partisans and both their opponents, both theories will be given.

(*a*) *Theory of Crile.*[3]—Fall in the arterial blood-pressure is the essential phenomenon according to Crile, and without this fall there is no surgical shock. The cause of the hypo-

[1] Archiv für Physiol. Abt., 1895, p. 200.
[2] Centralbl. für Physiol., 1894–95, viii, p. 769.
[3] Harvey Lecture, 1908.

tension is traumatism of the nerve tissues and also physic stimuli. There is very little difference between death from

FIG. 43



Fatal postoperative shock. (Cook and Briggs.)

hemorrhage and death from shock, both being due to failure of the circulation, producing certain degenerations of the central nervous system. It is assumed that the fall in

blood-pressure is mainly due to a functional impairment, a break down of the vasomotor centres, that the heart and bloodvessels themselves are only secondarily affected, principally by reason of the anemia of low blood-pressure; that the cause of the functional impairment or breakdown of the vasomotor centres is due in part to the effect of excessive afferent stimuli, and in part to the progressive anemia of these centres, there occurring a species of vicious circle. These shock producing afferent stimuli are but little influenced by general anesthesia, but are totally blocked by cocainization of their conducting paths.

In a later work[1] there is a résumé of his views regarding the importance of operating on the principle of anoci association, which means the avoidance, as much as possible, of psychic and traumatic insults. Harmful or nocuous associations are called "noci associations," and if all nocuous associations are removed, this state is designated "anoci associations."

Fig. 43, from Cook and Briggs, shows admirably the blood-pressure in fatal postoperative shock.

(*b*) *Theory of Henderson.*[2]—It is generally held that carbon dioxid is the normal stimulant of respiration and that when this is diminished, respiration ceases. Henderson believes that when death follows intense physical suffering, not complicated by hemorrhage, there are two principal stages. At first the excessive breathing diminishes the carbon dioxid content of the blood. If at any time after this condition of acapnea (diminished carbon dioxid content of the blood) has been induced, the pain is greatly diminished and the respiratory centre is thus allowed to relapse into a standstill, fatal *apnea vera* may occur. If on the other hand, the pain is sufficiently continuous to keep the respiratory centre continually excited, then apnea is prevented, and the condition of acapnea becomes more and more acute and general until the circulation fails, and the subject sinks into surgical shock. Both fatal apnea and the more

[1] Jour. Amer. Med. Assoc., 1912, lix, p. 114.
[2] Bull. Johns Hopkins Hospital, 1910, xxi, p. 235.

slowly developing failure of the circulation are due initially to acapnea induced by the excessive breathing occurring during torture.

Henderson believes that respiratory failure precedes low blood-pressure, and in endeavoring to repeat Crile's work, has become more convinced of this view. In fact, he claims that the majority of Crile's dogs had behaved as had his, and that Crile's experience had been the same as his own, though differently interpreted.

(*B*) **Hemorrhage.**—Hemorrhage can scarcely be classed as a surgical complication, for with the present surgical technique extensive bleeding rarely occurs. The effect of hemorrhage in surgery is precisely the same as that seen in typhoid (q. v.) and these effects cannot be more correctly gauged, both at the time of operation and afterward, than by frequent blood-pressure estimations.

(*C*) **Acute Dilatation of the Stomach.**—There is no mention in the literature of blood-pressure in this common surgical complication. In a case of dilatation in pneumonia, the writer found the blood-pressure to be 95 mm. Hg. just before symptoms of dilatation began, having fallen from 114 the night before. The blood-pressure in postoperative dilatation would doubtless be low, as there is considerable shock associated with this condition.

CHAPTER XII.

BLOOD-PRESSURE IN CERTAIN CONDITIONS OF THE GASTRO-INTESTINAL TRACT.

Intra-abdominal Pressure and Blood-pressure.—Experimentally, this question has been studied by Hamburger[1] and Qurin.[2] Hamburger used physiological salt solution to raise the pressure in the peritoneal cavity and found that up to a certain point, the arterial pressure ran equally with the abdominal pressure, but when the latter was further increased, there was a fall in the former. Qurin employed air, but arrived at the same results as did Hamburger five years previously, and both are agreed that the phenomenon is best explained as follows: By increasing the abdominal pressure there is brought about increased resistance in the veins owing to the compression of the latter. The heart is called upon to do increased work and draws upon its reserve energy. When the pressure in the abdomen is still further increased, the heart becomes exhausted and the blood-pressure falls. Although neither authority applies his observations to man, it is apparent to the clinician just what pernicious effects a large accumulation of fluid must have on the general circulation, especially when such accumulations are secondary to disease of the heart. Early release of ascitic fluid is an imperative indication in those cases where the heart, owing to the valvular or muscular inefficiency, is already laboring at a disadvantage. (See Chapter VIII, p. 146.)

A recent contribution to the subject is that of Fundner[3] who believes that the mechanical pressure on the diaphragm

[1] Archiv für Phys., 1896, Physiolog. Abt., p. 332.
[2] Deutsch. Archiv für klin. Med., 1901, lxxi, p. 79.
[3] Deutsch. med. Woch., 1913, p. 646.

has nothing to do with the increase of blood-pressure, but that the rise is due to reflex processes arising from the stomach.

Relaxed Abdominal Walls.—It is generally supposed, although not all writers are in accord, that blood-pressure rises when the subject assumes an upright posture and falls when he lies down. Birtch and Inman[1] having observed, in patients suffering with enteroptosis and relaxed abdominal musculature, that the pressure fell 10 to 25 mm. Hg. on standing after lying, undertook extended studies with this as a text. The severity of the symptoms bore some relation to the amount of the systolic fall, being more marked with a low reading. On the days when the individuals were feeling their best, the reading taken while standing remained close to the one obtained in the recumbent posture.

In cases of enteroptosis, as the table will show, there was a fall in systolic pressure from 10 to 25 mm. Hg. when the patient stood up, and the diastolic remained stationary or fell, decreasing the pulse pressure, as is seen in the healthy individual. They call attention to the fact that decrease in pulse pressure in persons with relaxed abdomens is caused by fall in systolic pressure, not by diastolic rise as is the case in health.

As a contrast to these cases, the authors offer a second table showing the effect on blood-pressure of postural change in nephritis. In these individuals there was a preservation of systolic and diastolic pressures as is seen in athletic youths. The accompanying neuropathic symptoms of visceroptosis are believed by Birtch and Inman to be due to the cerebral anemia and not to the malposition of the viscera, the cerebral anemia, in turn, being due to the muscular atony disturbing the splanchnic circulation.

[1] Jour. Amer. Med. Assoc., 1912, lviii, p. 265.

Type of Cases of Relaxation of the Abdominal Wall.

Subject.	Time.	Position.	Maximum.	Minimum.	Pulse pressure.	Pulse rate.
1	2.00	R.	118	85	33	76
1	2.15	S.	92	72	20	92
2	2.46	R.	124	85	39	84
2	2.55	S.	104	80	24	92
3	2.30	R.	130	95	35	84
3	2.40	S.	110	85	25	104
4	3.33	R.	110	78	32	70
4	3.45	S.	100	70	30	90
5	2.10	R.	120	95	25	84
5	5.20	S.	110	95	15	90

Reclining (R.). Standing (S.).

Average of Twenty-five Cases.

Fall of systolic	19.5 mm.
Fall of diastolic	6.1 mm.
Fall of pulse-pressure	8.0 mm.
Increase in pulse-rate	15.0 mm.

Relaxed Musculature Complicated with Chronic Nephritis.

Subject.	Time.	Position.	Maximum.	Minimum.	Pulse pressure.	Pulse rate.
1	2.15	R.	150	95	55	72
1	2.25	S.	158	110	48	76
2	11.05	R.	165	110	55	68
2	11.15	S.	165	110	55	74
3	11.30	R.	175	90	85	64
3	11.45	S.	165	90	75	66
4	12.14	R.	180	95	90	78
4	12.25	S.	174	110	64	90

Reclining (R.). Standing (S.).

Average fall of systolic	2.00 mm.
Average increase of diastolic	7.50 mm.
Average fall of pulse pressure	10.75 mm.
Average increase in pulse rate	6.00 mm.

CHAPTER XIII.

BLOOD-PRESSURE IN DISEASES OF THE INTERNAL SECRETORY GLANDS.

I. DISEASES OF THE THYROID.

Graves' Disease.—The only publications devoted specifically to the study of blood-pressure in Graves' disease are those of Maire,[1] Treves,[2] and Spiethoff,[3] the last named using the instruments of Riva-Rocci and von Recklinghausen. The general opinion is that the blood-pressure is high, although this has been arrived at mostly by guesswork and not from actual experience.

Spiethoff has made a thorough study of the pressure in 20 patients, but found no constant relation between blood-pressure and severity of the illness. In patients with low blood-pressure (120 to 127 R.R.), the subjective symptoms were just as pronounced as in those with high pressure, (153 to 159 mm. Hg., R.R.). He concludes that the pressure is not uniform in the disease, and says that statements to the effect that blood-pressure is always high or always low, are incorrect. High and low pressures are met alike in severe cases of Graves' disease, while in the milder forms, normal pressures are the rule. This variability is believed to be, in the main, cardiac in origin, although vasomotor influences can not be excluded.

Myxedema.—Gordon believes the blood-pressure is normal in this condition.

[1] Thèse de Paris, 1883.
[2] Riv. iconogr. del policlin. gen. di Torino, 1897, i, p. 2.
[3] Centralbl. für inn. Med., 1902, xxiii, p. 849.

II. DISEASES OF THE ADRENAL GLAND.

(1) **Acute Adrenal Insufficiency.**—This comparatively rare condition, rare presumably because so infrequently recognized, has been rather cavalierly treated by recent writers, so far as blood-pressure observations are concerned. Boussuet claims to have diagnosed acute insufficiency eight times by this symptom complex: asthenia, hypotension, nausea, vomiting, and diarrhea. The symptoms all subsided after hypodermics of adrenalin. Lavenson[1] has made a careful review of the subject in connection with the report of a case which he had observed, but unfortunately there were no blood-pressure estimations although the report of the examination seems to indicate vasomotor relaxation.

A few years later, Cooke[2] reported a case, also without blood-pressure estimations. He made the very interesting and extremely valuable observation that solution of the diseased portions of the adrenals obtained at autopsy produced no rise in blood-pressure in a dog, although the healthy portions of the same glands had pressor properties. That low blood-pressure must be a common feature, though not often reported, is apparent from these studies, and that the hypotension is in part due to functional deficiency of the adrenal seems also certain, and receives confirmation from Boussuet's findings.

(2) **Addison's Disease.**—Hypotension is a constant feature in Addison's disease.

Dock[3] reports four cases with pressures of 110 to 85, 108 to 75, 85 to 65, and 82 to 60. The last two cases were seen only a few days before death.

III. DISEASE OF THE PANCREAS.

Diabetes Mellitus.—One would expect to find cardiovascular derangements in diabetes as in other toxic states.

[1] Archiv. Int. Med., 1908, ii, p. 62.
[2] Ibid., 1912, ix, p. 108.
[3] Modern Medicine, Osler, vi, p. 368.

A study of total statistics shows that approximately 17 per cent. of all diabetics have some cardiac degeneration, especially hypertrophy, but other changes are not infrequently observed, *i. e.*, fatty myocardial changes. The first studies from the stand-point of blood-pressure, are those of Potain, who found it to be very high, higher even than that seen in nephritis. This view is shared by Tiessier,[1] while conflicting views are those of Vaquez, Hensen, and Ott, who all report cases showing hypotension. The writer has seen both high and low pressures, but low pressure more frequently.

There is apparently no relation between the amount of sugar and degree of blood-pressure, except it be an inverse proportion. Acidosis seems to lower the systolic pressure, for in 10 cases, with an average age of thirty-three, the blood-pressure averaged 107 mm.; while in 15 cases with no sign of acid intoxication and an average age of fifty-three, the systolic pressure averaged 140 mm (Elliott[2]).

Complications are probably at the bottom of the hypertension, and only those cases which have nephritis or arteriosclerosis exhibit this phenomenon. A pure uncomplicated diabetes, irrespective of the amount of glycemia, is not accompanied by increase of blood-pressure.

Ehrmann[3] finds that some time before coma appears there is a marked decrease in blood-pressure, and he regards this as the cause of the diabetic collapse.

IV. DISEASE OF THE PITUITARY.

Acromegaly.—Gordon reports two cases with pressures of 110 and 90 respectively. Dr. Mark, in his extraordinary "Acromegaly" (a personal experience) unfortunately omits mentions of sphygmomanometric records.

In a boy, recently under my care, who exhibited signs of disease of the posterior portion of the gland, there was hypotension.

[1] Gaz. des Hôp., November 8, 1904.
[2] Jour. Amer. Med. Assoc., 1907, xlix, p. 27.
[3] Berlin. klin. Woch., 1913, p. 13.

CHAPTER XIV.

BLOOD-PRESSURE IN OPHTHALMOLOGY.

In cases of anemia, where there is usually low arterial blood-pressure, the veins of the retina are seen to pulsate, and this has been held to indicate a lowering of venous pressure. Cases of increased venous pressure are sometimes associated with dilatation and tortuosity of the retinal veins. Increase of pressure is generally found in venous thrombosis of the vessels of the eye and brain, and in tumors pressing on the eye-ball, although the increase is but temporary.

As far as increased *arterial* tension is concerned, the eye changes are not at all characteristic and cannot be differentiated from changes due to angiosclerosis. Indeed, the changes in the retina may precede and antedate any sphygmomanometric hypertension, and may occur independently of any general increased blood-pressure. The converse is also true, that hypertension, apart from arteriosclerosis, can occur without any opthalmoscopic changes in the eye.

de Schweinitz[1] calls attention to pathological alterations in the eye-ground when arteriosclerosis is the cause of the hypertension, dividing the changes into those which are suggestive and those which are pathognomonic.

Of the suggestive signs, importance is laid on uneven calibre of the retinal vessels, and disturbance of the central light streak and an unusually light color of the breadth of the artery. The pathognomonic signs are changes in the size and breadth of the retinal arteries, giving a beaded appearance with a distinct loss of translucency. Perivasculitis, in the form of white stripes in the arteries, with alternate contractions and dilatations of the veins, and of

[1] Trans. Amer. Ophthal. Soc., 1906–08, xi, p. 87.

most importance, indentation of the veins by the hardened arteries, are all valuable signs. As the condition progresses gray opacities around the disk or following the course of the vessels (edema) and hemorrhagic infiltrations or extravasations are seen.

The eye changes are held to be of great importance in the diagnosis of arteriosclerotic hypertension, as these alterations are found in no other condition. (See Arteriosclerosis.) Just how early these signs appear is not definitely known, but it would seem that they are exhibited early enough to render an opthalmoscopic examination a very important aid in the diagnosis of arteriosclerosis. There is no mention of eye changes in cases of arteriosclerosis with normal or lowered blood-pressures. Were these changes to be noted, the eye examination would take precedence over any means at our disposal for diagnosing arteriosclerosis. Although it is of scientific interest and may be of clinical value to know definitely the condition of the blood-pressure in such states, it is far more important to know the eye changes in every case of hypertension.

In estimating blood-pressure, one should never forget its lability, and should estimate values obtained only after the incidence of age, sex, work, mental state, etc., is taken into careful consideration. Since the blood-pressure instrument is now so firmly established as a necessary detail of the physician's armamentarium, it may not be too optimistic to hope that with the early recognition of hypertension and with treatment directed to its lowering, many cases of irrevocably impaired vision may be prevented.

Optic nerve atrophy and retinal changes are commonly seen after severe hemorrhages, in which condition there is generally lowered blood-pressure. Cases of so-called progressive pernicious essential anemia with low pressure develop massive retinal hemorrhages, and this finding is not unusual in cases of secondary anemias of severe grades.

Primary glaucoma is generally associated with hypertension. Löhlein[1] has devoted a special study to the cause

[1] Archiv für Ophthal., 1912, lxxxiii, p. 547.

of increased arterial pressure in glaucoma and discards as worthless the theory that adrenalin is the cause of the high blood-pressure. Certain of his cases had low pressure, 105 mm. Hg. Unfortunately Löhlein does not state which instrument he used for his work.

The eye change, seen in nephritis or in other conditions of increased intracranial pressure, are well known. Opthalmologists seem to be united in the belief that retinal hemorrhages are a direct result of increased arterial pressure, which does not in the least alter the apparently parodoxical statement that low pressure also seems to favor intraocular extravasations.

From the stand-point of the ophthalmologist then, the estimation of blood-pressure is most important, as cases of retinal hemorrhages due to lowered pressure must be differently handled from those arising from hypertension. In the chapter devoted to treatment of hypertension, the great value of the sphygmomanometer in guiding the course of treatment will be discussed, but the point must be emphasized here, that there is in all cases of hypertension, a readjustment of physiological balance whereby the individual's standard exceeds 130 mm. Hg.—the normal systolic pressure (auscultatory). His normal limit may rise to 140 or 150 mm. Hg., and one should have continually before him the knowledge that, to reduce a pressure beyond the new limit which Nature has created for its own defense, is a malicious intrusion on her domain, and may be followed by dire consequences. No treatment directed at hypertension should ever be instituted without careful control of therapy by painstaking use of the sphygmomanometer.

CHAPTER XV.

EFFECT OF DRUGS AND OTHER THERAPEUTIC MEASURES ON BLOOD-PRESSURE.

THIS chapter must in no way be considered a treatise on pharmacology and physical therapeutics. The endeavor has been in these pages, merely to present evidence concerning the action of certain therapeutic measures and drugs, which evidence, in part, at least, has led to the generally accepted opinions regarding their behavior in cases of hypertension and hypotension.

I. HYPERTENSION.

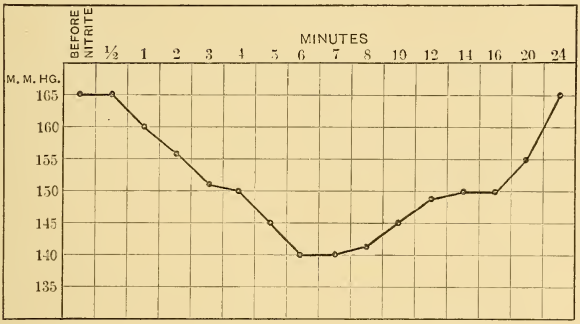
1. Drugs of the Nitrite Group.—(*a*) **Nitroglycerin.**—The effect of this drug was *nil* when given in tabloid form (Matthew), and the only preparation which yielded any results was the Solution trinitrini.

The vasodilator effect followed within a minute after administration, and the reduction in pressure amounted to 25 to 30 mm. Hg. The maximum effect is produced in five minutes, and is maintained for two minutes, after which time the pressure begins to rise again, reaching its original level in a half-hour. Repeated doses had no effect in reducing permanently the pressure. Matthew found a tolerance could be acquired, so that 4 c.c. (60 minims) a day were readily borne, but he calls attention to the possibility of doing harm by such doses. Headache and shortness of breath were early symptoms of toxicity both of which disappeared with the cessation of the nitrite.

(*b*) **Nitrites.**—There is a vasodilator effect after the administration of nitrites which is produced usually in five minutes

(Matthew), but the maximum effect (fall of pressure 32 mm.) is seen in fifteen minutes. The maximum fall is maintained

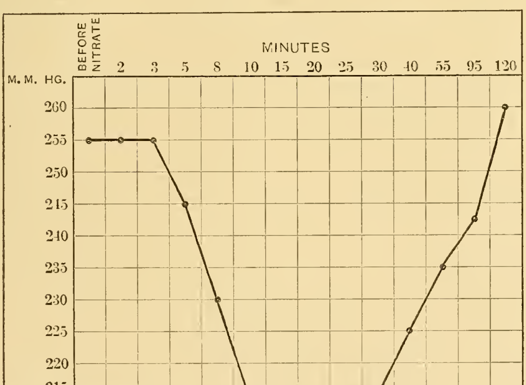
Fig. 44



0.13 c.c. (two minims) liquor trinitrini. (Matthew.)

for from twenty to sixty minutes, the original pressure being again reached in one to two hours. Repeated doses of 0.13

Fig. 45



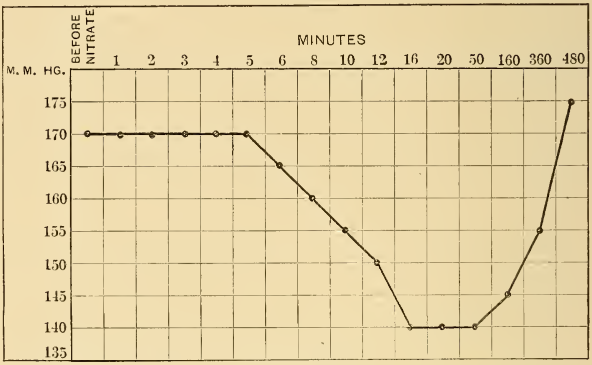
0.13 (two grains) sodium nitrite. (Matthew.)

(2 grains) of sodium nitrite three times a day, kept the blood-pressure down, not constantly at the lowest level of a single

dose, but still quite low, and after prolonged medication the pressure rose, within twenty-four hours, to its normal level, when the nitrite was discontinued.

(*c*) **Erythrol Tetranitrate.**—The effect is evident in about five minutes, the fall of pressure being gradual and amounting to 35 mm. Hg. In about twenty-two minutes, the maximum effect is produced, which is maintained for about two hours, after which the rise toward the normal level sets in, the complete effect passing off in from five to six hours. There is no tolerance to this drug which is not so well borne as some of the other drugs.

Fig. 46



0.06 (one grain) erythrol tetranitrate. (Matthew.)

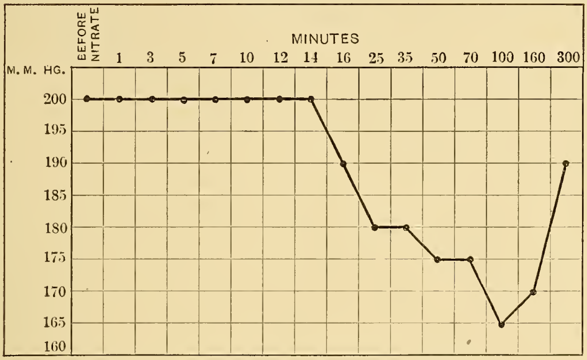
(*d*) **Mannitol Nitrate.**—In about fifteen minutes a fall of pressure begins, which reaches its maximum in two and a half to three hours (35 mm. Hg.). After three hours, the pressure begins to rise, reaching normal in about six hours.

Matthew gives the following practical hints in prescribing the foregoing vasodilator drugs:

1. Liquor trinitrini. Where a single dose is prescribed, two minims is the one most likely to obtain the desired effect.

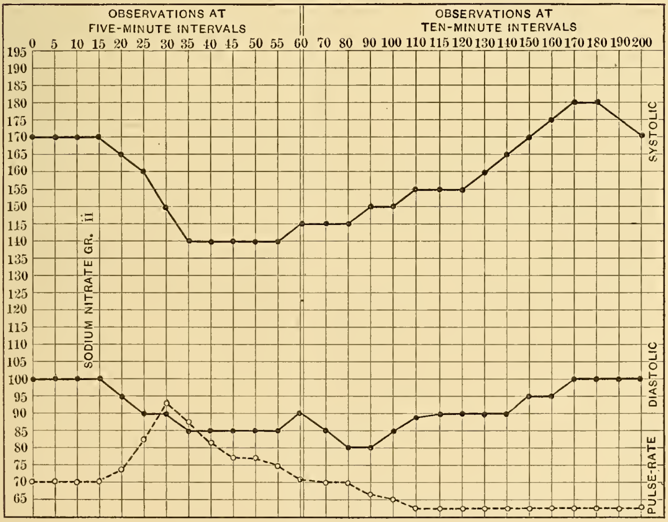
2. Sodium and potassium nitrite. In suitable nitrite cases, 0.13 (2 grains) produce a reduction of just over 30 mm. Hg. This action will last two hours and only after this is it

FIG. 47



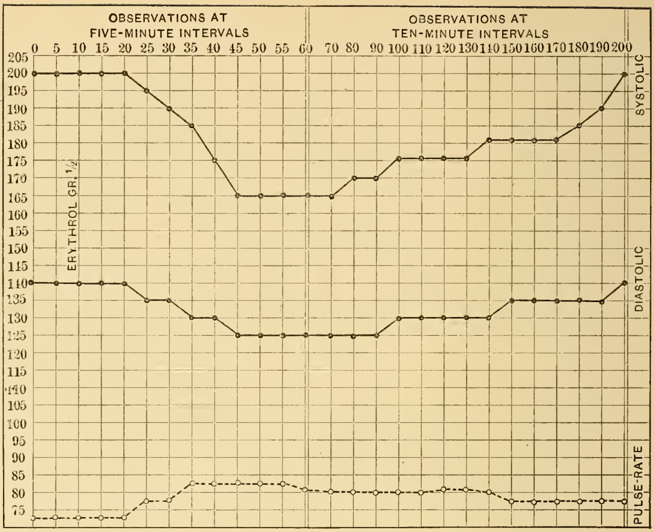
0.06 (one grain) mannitol nitrate. (Matthew.)

FIG. 48



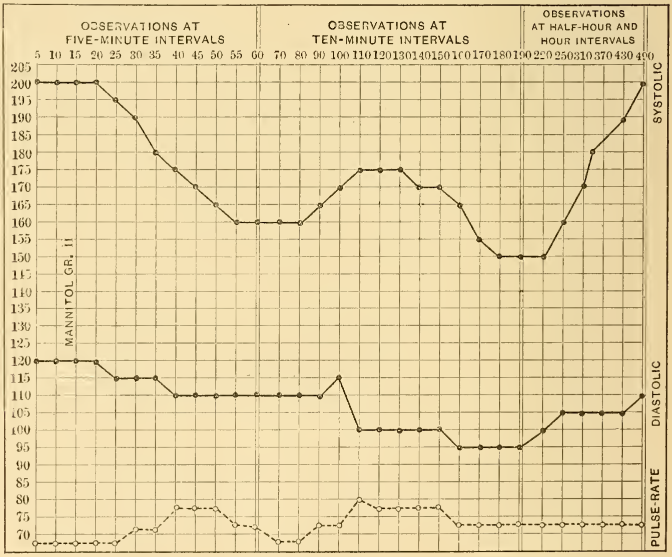
Curve obtained by the use of sodium nitrite. The acceleration of the pulse rate during the fall in pressure is the rule; slowing of the pulse at that point was observed in a few cases. The systolic rise above the original level at the end of the curve is more common with this drug than with those causing a more gradual curve. No terminal diastolic rise above the original level was noted. Dastolic fall half the systolic. (Lawrence.)

FIG. 49



Curve characteristic of the action of erythrol. Less acceleration of pulse than with sodium nitrate. Diastolic fall very slight—less than half systolic. (Lawrence.)

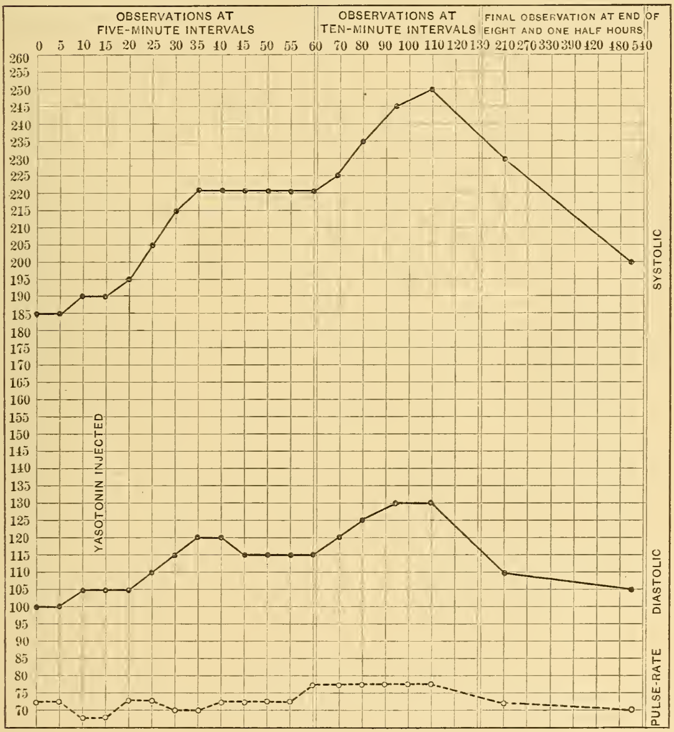
FIG. 50



Effect of mannitol. Acceleration of pulse slight. Diastolic fall slower than systolic and but slightly more than half as great. Diastolic rise slower than systolic, resulting in increased blood-pressure at end of curve. (Lawrence.)

necessary to repeat it. No benefit is obtained by increasing the dose, and a small dose will not give the desired effect.

Fig. 51



Effect of vasotonin. A considerable and prolonged rise in both systolic and diastolic pressure. Pulse practically unaffected. The patient was suffering from arteriosclerosis. His kidneys were apparently only slightly affected. (Lawrence.)

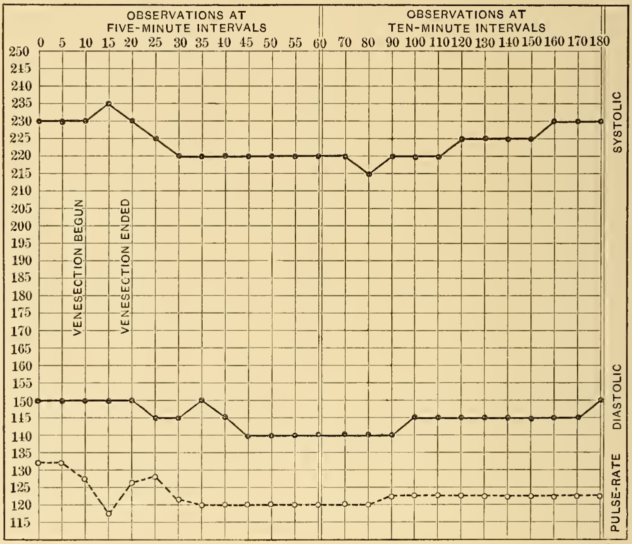
3. Erythrol tetranitrate. With this drug, a dose of 0.03 ($\frac{1}{2}$) to 0.06 (1) grain will produce a beneficial reduction, and the effect will last about six hours. Matthew recommends it to be used in all cases to start with.

4. Mannitol nitrate. Doses of 0.06 (1) grain are useful,

and there is no individual susceptibility to the drug or any tendency to unpleasant effects from its use.

In writing on the effect of *pressure-lowering drugs* and of *therapeutic measures* on systolic and diastolic pressure in man, Lawrence[1] makes some interesting contributions

Fig. 52



Effect of venesection. Action in this case less marked and less sustained than in others. Patient uremic and moribund. Pulse slowed. Diastolic fall two-thirds as great as systolic, therefore pulse pressure diminished less than by vasodilators. (Lawrence.)

to our knowledge of such therapeutic agents. Observations were made with the Faught apparatus 12 cm. cuff, the systolic pressure being estimated by palpation and the diastolic by the auscultatory method. The drugs used were sodium nitrite, erythrol tetranitrate, mannitol hexa nitrate and vasotonin.

The other therapeutic measures employed included

[1] Archiv. Int. Med., 1912, ix, p. 409.

venesection, hot-air baths, electric-light baths, and high frequency currents. Venesection reduced the pressure more persistently than any measure, in many cases the pressure did not regain its original level in twelve hours, and in another case not for thirty-six hours. Pressure was more frequently lowered after electric-light or hot-air baths than with the high frequency current. (Compare these charts with Matthews.)

The practical conclusions are that sodium nitrite reduces diastolic pressure more rapidly than the more complex compounds, but that none of the nitrite group is efficient for maintaining pressure at a permanently lowered level, as a tolerance is soon acquired. If the dose be still further increased unpleasant symptoms result.

Table I.—Average Blood-pressure Results from Administration of Nitroglycerin, Sodium Nitrite and Erythrol Tetranitrate to Normal Persons.

Drug.	Beginning action, min.	Maximum affect, min.	Duration of action, min.	Maximum extent of action.	
				Mm. Hg.	Per cent.
Amyl nitrite, 3 minims	1	3	7	15	11
Nitroglycerin, 1.5 mm., 1 per cent. solution	2	8	30	15	11
Sodium nitrite, 1 gr.	10	25	60	14	13
Erythrol tetranitrate, 0.5 gr.	15	32	120–240	16	14

Table II.—Average Blood-pressure Results from Administration of Nitroglycerin, Sodium Nitrite and Erythrol Tetranitrate to Patients with Arteriosclerosis.

Drug.	Beginning action, min.	Maximum affect, min.	Duration action, min.	Maximum extent of action.	
				Mm. Hg.	Per cent.
Nitroglycerin $\frac{1}{30}$ gr.	2	8	35	32	17
Sodium nitrite, 2 gr.	15	45	120	53	25
Erythrol tetranitrate, 2 gr.	30	60	180	60	30

Venesection has a more lasting effect than any drug, the diastolic pressure remaining depressed much longer than the systolic pressure, thus lengthening the pulse pressure.

The effects of hot-air baths, electric-light baths, high frequency current, and vasotonin are all uncertain, and the last named is unsafe and should never be used.

The two tables on page 197 illustrate Wallace and Ringer's results with drugs of the nitrite group[1] given to normal and diseased individuals:

2. **Iodids.**—When given to man, iodids have little effect in hypertension when the latter is due to nephritis as is so frequently the case. The good effects seen sometimes in arteriosclerosis may be due to the action on an underlying infection (syphilis) and not to any action on the arteriosclerosis process itself.

Intravenous injections seem to raise the blood-pressure in animals.[2]

Ghelfi[3] administered potassium iodid in increasing amounts to certain patients, and found at the beginning of the "cure" there was a rise of pressure of 30 to 40 mm., in some cases, and a fall in others. Continuance of the large doses of iodids ultimately leads to fall of blood-pressure with rapid pulse and vasodilatation.

Matthew[4] believes that iodids have a typical vasodilator effect on the peripheral arterioles, acting in a way similar to nitrites, not with the latter's celerity of action, but with a much more prolonged effect. A single dose, however, is not followed by vasodilatation, but if repeated three or four times a day, and continued thus, an effect is manifest within thirty hours. Indication for the use of iodids is high blood-pressure without arteriosclerosis, but in advanced arteriosclerosis they have no hypotensive action. Organic iodids contain too little iodin to be efficient. Sajodin is to be used only when alimentary disorders contra-indicate the use of iodids.

[1] Jour. Amer. Med. Assoc., 1909, liii, p. 1629.
[2] Barbiera, Arch. für d. ges. Phys., 1900, lxxix, p. 312.
[3] Abstr. in Münch. med. Woch., 1906, p. 2216.
[4] Edin. Med. Jour., 1911, i, p. 228

3. **Veratrum Viride.**—Pesci[1] recommends the giving of 20 to 30 drops of the fluidextract in all cases of hypertension in which the arteries still retain their elasticity. The best results with the drug are obtained in chronic nephritis, in which condition, the author says, it wards off uremia, and also tends to lessen the phenomena of the latter when once established.

In vascular spasm of the abdominal vessels, as is found in chronic lead intoxications the drug is useful and also when such spasm is a part of general arteriosclerosis, the march of the latter process is said to be inhibited by the use of veratrum viride.

The author cannot share the enthusiam which certain writers have for veratrum viride, and he has seen no good effects from its use.

4. **Effect of Carbon Dioxid Baths on Blood-pressure.**—If one reads the earlier works on the effect of carbonic acid baths on blood-pressure, he will find the opinion universally expressed that carbon dioxid raises the blood-pressure and for that reason they should not be used in arteriosclerosis. The Groedels[2] have shown that there is a slight rise in pressure on first getting into the bath, but it is no more of an elevation than one sees after many physiological acts. There follows very shortly a fall, which persists during the bath, but when the patient is removed from the bath, the pressure returns to normal.

Laqueur[3] reports the results obtained in 100 patients, as far as blood-pressure, pulse pressure and pulse rate are concerned. He employed the apparatus of Fischer and Kiefer, using a mixture of carbon dioxid in cold water. Using a bath of 34° C. (93.2° F.) temperature, the systolic pressure was raised in 68.6 per cent. of the cases, although this increase was generally 2 to 5 mm. Hg., and in one case 23 mm. Hg. In 22.5 per cent. of the cases the pressure was lowered, and in the other cases it was unaffected. The pulse pressure was increased in 51 per cent. of the cases. The rise in

[1] Abstr. in Münch med. Woch., 1907, p. 281.

[2] Deutsch. med. Woch., 1906, p. 1371.

[3] Ztsch. für exper. Path. und Therap., 1909, vi, p. 855.

pressure is more marked in subjects with a functionally normal cardiovascular apparatus, more so than with baths of the same temperature, but containing no carbon dioxid. Oxygen baths have no such blood-pressure raising effect, due to the absence of cutaneous stimulation which arises from the carbon dioxid bath. Laqueur does not share the view of Senator, that the effect of carbon dioxid baths comes from the contrast of the cool water and the bubbles which give the sensation of warmth, but believes that there is some property in the carbon dioxid itself.

In baths of 33° C. (91.4 F.) and below this temperature, the systolic pressure was found raised in 77 per cent. the pulse pressure was increased in 51 per cent. and the pulse rate diminished in 78.7 per cent. In cases of contracted kidneys and arteriosclerosis with hypertension, there is no decrease in blood-pressure or pulse pressure, although cases with "présclèrose" often show a moderate fall.

Newton[1] is much more enthusiastic than Laqueur regarding the benefit of the Nauheim baths in cases of hypertension arising from nephritis, although he is uninformed as to their *modus operandi*. He leans to the belief that increased elimination through the skin is a not inconsiderable factor.

A comprehensive study of the effect of Nauheim baths has been made by Swan[2] with results which differ materially from the above quoted, although there is no explanation offered for such contradictory opinions.

There is, according to him, no constancy of action on the blood-pressure of carbonated brine baths, and there is no method of determining in advance whether a given treatment will be followed by an elevation or by a fall of pressure. The tendency of the bath is toward elevation, however, as the systolic pressure was raised more frequently than it was lowered. There are some cases of hypertension in which carbonated brine baths have produced a lowered systolic pressure, and there are others in which the ultimate pressure has been higher than it was at the beginning.

[1] Amer. Jour. Med. Sci., 1912, cxliii, p. 578.
[2] Arch. Int. Med., 1912, x, p. 73.

5. **Oxygen Baths.**—In normal subjects there occurred practically no reduction in blood-pressure from the oxygen bath, but in most organic heart affections when compensation is fairly well established, the blood-pressure is almost unfailingly reduced.[1] Sadler believes that early arteriosclerosis (présclèrosis) the effect of the baths is extremely beneficial, producing in one case a fall in blood-pressure of 35 mm., but in advanced arteriosclerosis the results are disappointing. In cases of functional hypertension, with no discoverable cause of the high blood-pressure, the baths have been found to have great value. The great contra-indication to their use is cardiac decompensation.

Best results are obtained with a temperature of 34° C. (93.2° F.) to 35° C. (95° F.), but in certain nervous cases 36° C. (97°) F. has been the temperature employed. The bath should not be given within an hour of bedtime.

6. **Carlsbad Cure.**—Ritter[2] found a lowering of blood-pressure during Carlsbad cure.

7. **High Frequency Currents.**—This form of current was demonstrated in 1891, by Nicola Tesla, being a current in which cycles of positive and negative waves exceed 10,000 per second.

As used in medicine, the frequency runs much higher then this low limit; the cycles ranging from 200,000 to 2,000,000 per second, and with the tremendous voltage of from 10,000 to 500,000 volts.

Both frequencies and voltages much in excess of these limits, may freely be passed through the system, but when given much higher than the limits mentioned, they gradually lose their therapeutic properties on the human system.[3]

Van Rensselaer believes that the underlying causes of most cases of hypertension are metabolic, and that the high frequency current by improving the latter processes, reduces the high pressure. The current also checks the advance of the pathological processes seen in renal or cardiac disease.

[1] Sadler, Amer. Jour. Physiol. Therap., 1910–11, i, p. 417.

[2] Deutsch. Arch. für klin. Med., 1910, c, p. 11.

[3] Van Rensselaer, Albany Med. Jour., 1913, p. 77.

Sayer[1] uses the current in doses of 0.028 to 3 units for six minutes, and believes a rise in pressure is apt to follow if stronger currents are employed. Individuals with low blood-pressure find the treatment very disagreeable. The mode of action of the currents is purely hypothetical. Bruce[2] publishes a report of a patient who died during treatment by high frequency current.

8. **Use of Diet in Hypertension.**—The cardinal principles of a dietary directed toward the amelioration or cure of hypertension is detailed very clearly by Hecht.[3] The requisites of the diet are:

1. "Spiceless" food.
2. Lessening of the meat intake.
3. Increase of vegetable diet with increase of mineral intake with the food.
4. Lessening of fluid intake.
5. Lessening of salt.
6. Lessening or even cessation of alcohol.

1. Such things as pepper, paprika, mustard, which are renal irritants, should be avoided. To make the food appetizing or rather to gain an appetite for the insipid tasting food, it is recommended to give 15 drops of the compound tincture of cinchona before each meal, and it is sometimes of value to insert a hunger day (greatly reduced amount of food) with rest in bed, as a stimulant to a jaded appetite. This spiceless food seems to have in the over-fed, corpulent type of individual, a very useful application, on account of its appetite decreasing properties, which tend to lessen the desire for alcohol and gourmandism.

2. By lessening the meat intake, two results are obtained—a cutting down of the excessive protein intake and a great reduction of the so-called "extractives." The majority of individuals eat too much, and more people die of over-eating than of overdrinking. Voit has placed the lowest protein requirement at 1.5 grams per kilogram body weight, but Chittenden's experiments have shown the obvious

[1] Brit. Med. Jour., 1910, ii, p. 1052.
[2] Medical Electrol. and Radiol., 1905, vi, p. 231.
[3] Ztsch. für klin. Med., 1912, lxxvi, p. 87.

fallacy of this, as he has been able to keep hard-working men in nitrogen balance on food containing but 0.7 to 0.9 grams per kilogram body weight. As substitutes for meat, milk and cheese may be used. There should be no distinction made between light or dark meat. The latter is, to be sure, freer of extractives, but these can be removed by boiling.

3. Vegetable diet. In cases of uncomplicated non-albuminous hypertension (présclèrose) the blood-pressure can be brought to normal in a few days on purely vegetable food. In nephritis, such results are never seen, although a lowering of pressure is seldom missed. In cases of neurasthenia, with depression, a vegetable diet is to be especially recommended. When there is malnutrition associated with the high blood-pressure, a carbohydrate rich food is especially useful. Vegetables such as spinach, peas, cabbage, cauliflower, Brusselssprouts, beans, carrots, beets, potatoes and cereals are recommended. With the meat there should be two vegetables, one boiled and the other rice or potatoes in various forms. Four evenings a week it may be advisable to have a pure vegetable day or a cereal day. Salad (no vinegar) and radishes (the latter only when there is no gastro-intestinal disease or nephritis) may be freely used. Fresh or stewed fruits are especially valuable.

When obesity is combined with hypertension, fats and carbohydrates must be restricted, especially cereals, bread and potatoes. The latter may be eaten twice a week, and bread to the extent of 20 to 40 grams, given as toast or zwiebach. Rice (30 grams raw or 80 grams boiled) once a day. Fatty food is best avoided, but a small amount of butter 10 to 15 grams in the morning) may be given. Tea, coffee, and stewed fruit are best taken without sugar. Associated with properly regulated passive or active massage, both hypertension and obesity can be very much benefited.

4. Fluid intake. If fruit, compote, vegetables and salads are eaten, water with meals may be dispensed with, since the non-stimulating diet does not produce excessive thirst. Caffein-free coffee and very weak tea, both not too hot, may be induced in very small amounts.

5. Salt intake. It is questionable if sodium chlorid has, of itself, any blood-pressure raising properties, but since it causes thirst, necessitating the use of much water which does seem to raise pressure, a salt-poor diet is advisable.

6. Alcohol. Although we have learned that alcohol, *per se,* does not raise blood-pressure it has an injurious effect on the bloodvessels and in this way tends to raise pressure.

Food should be well chewed, the intervals between meals should not be too long as they lead to weakness and to intense thirst and hunger.

II. HYPOTENSION.

1. **Digitalis.**—Hypodermic injections of digitalin act more promptly and more energetically than strychnin, but its effects are not so permanent. A combination of the two drugs is of service when strychnin alone fails to raise the pressure.

Tincture of digitalis in cases of hypotension and hypertension produced no blood-pressure rise (Tinct. digitalis ♏xv every four hours) (Burnet), while others claim that it sometimes raises, sometimes lowers, and sometimes does not change the blood-pressure, that is to say, the maximal pressure. It does, however, always lower the diastolic pressure thereby increasing the pulse pressure.

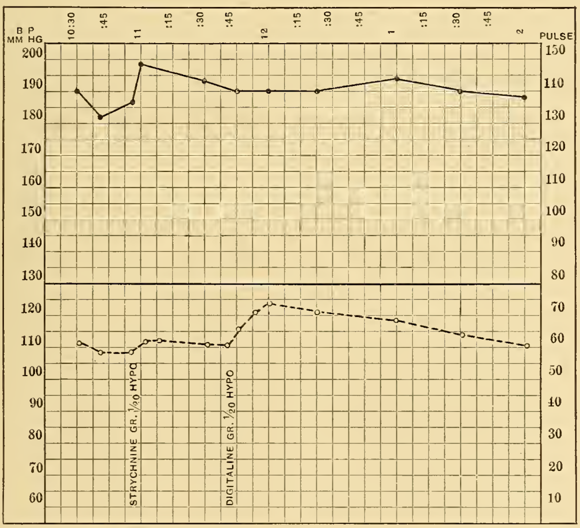
Price[1] discredits analogies being drawn between animal experimentation and observations in man, concerning the action of digitalis. In the former, very large doses can be studied, which is impossible in man. He studied (Riva-Rocci) the effect of ʒj of the tincture a day, and found in twenty-one cases, that there was a rise in pressure in only one case, in fact, in several cases there was a fall, and he believes digitalis *does not* raise the human pressure by constricting the peripheral vessels. He says if this conclusion is correct, then there is no contra-indication to

[1] Brit. Med. Jour. 1912, ii, p. 689.

its use in excessive hypertension and in cases of excessive disease of the arterial walls.

In some cases, dilatation of the heart may be due to increased arterial tension and digitalis in these cases, by improving the blood-supply of the brain, lessens the activity of the vasoconstrictor centre and a lower arterial tension often follows. Digitalis itself is without effect in raising blood-pressure.

Fig. 53

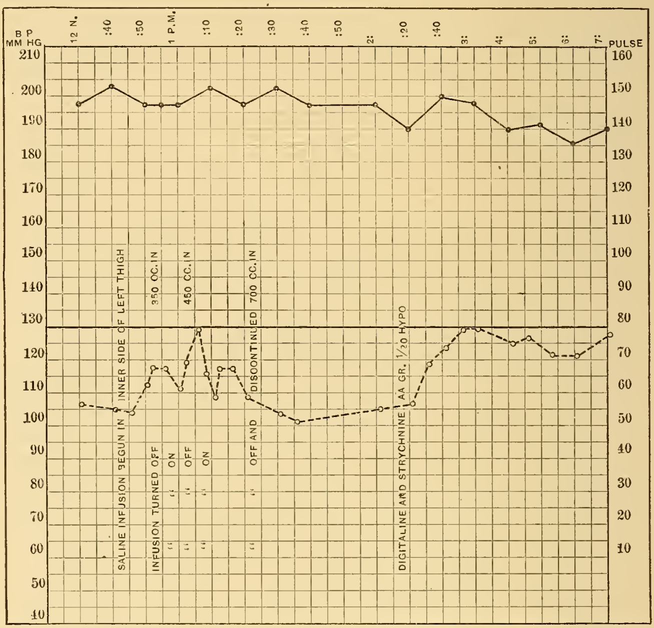


Fatal relapse in typhoid fever. Profound toxemia, showing effect of digitalin. (Cook and Briggs.)

2. **Strychnin.**—When strychnin is given in doses of 0.001 to 0.006 gram there is a rise in blood-pressure which is not so prompt as that induced by alcohol, but is more permanent (one to four hours). The drug when continued for eight or twelve doses seems to be without pressor effect, but if omitted for one or two doses, the action will again be seen. "On the whole, strychnin is by far the most satisfactory cardiovascular stimulant for long-continued routine administration, the maintenance of a satisfactory blood-pressure level, free

from intervals of depression, being most easily accomplished by its use in appropriate doses." (Cook and Briggs, see Fig. 55.)

Fig. 54



Profound typhoid toxemia, showing effect of digitalin and strychnin. (Cook and Briggs.)

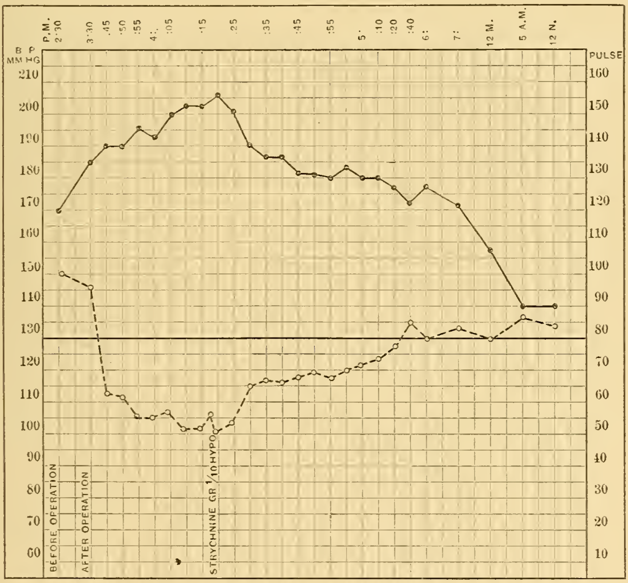
In some cases strychnin seems to be useless. These are moribund individuals with a circulatory apparatus rapidly failing, and in such a condition further forcing is hopelessly contra-indicated. A second class of cases includes those individuals with healthy tone of heart and vessels.

3. **Alcohol.**—The only indication for the use of alcohol, is toxemia. As a cardiac stimulant or as a vasomotor constrictor it is absolutely inert and any rise in blood-pressure which follows its use is wholly transient and results from a reflex action through irritation of the afferent nerves in the mucous membrane or in the skin, when administered hypodermically. It has no more vasomotor action than

has other irritants like tincture of capsicum. (Cook and Briggs.) (See Influence of Alcohol on Blood-pressure, Chapter V.)

4. **Camphor.**—Although extolled by continental writers as a valuable stimulant, its worth seems to have been held *sub judice* by American clinicians. Cook and Briggs point out that as a pressor drug it is absolutely inert in cases of

FIG. 55



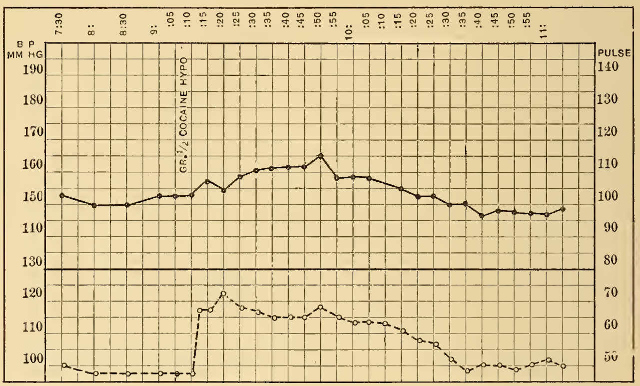
Postoperative depression, showing effect of strychnin. (Cook and Briggs.)

long-continued depression, as in typhoid fever. In cases of cardiac decompensation with hypotension, the writer is in accord with their observations that it has distinct merit in doses of 10 to 20 minims of a 10 per cent. solution. The indication for its use is well covered by digitalin, especially when the latter is used in combination with strychnin.

5. **Cocain.**—Cocain has a distinct pressor effect and must be classed as a true stimulant. Used with strychnin and digitalin in cases of postoperative or posthemorrhagic

hypotension it possesses decided value. (Cook and Briggs.) The blood-pressure has been seen to rise 20 mm. Hg., after 0.015 or 0.03 grain, and they recommend its use with saline injections, giving to the latter a "positive stimulant value which they do not otherwise possess." (Fig. 56.)

FIG. 56



Postoperative depression, showing effect of cocain. (Cook and Briggs.)

6. **Strophanthus.**—In some cases it produces rise in pressure.
7. **Squill.**—Uncertain in blood-pressure raising action.
8. **Caffein.**—Raises pressure.
9. **Pituitrin.**—The watery extract of the infundibular portion of the beef hypophysis seems to have, if authors are to be believed, wonderful properties. It is said to have a marked influence on metabolism, and to inhibit the growth of bone, although these are less important than its action on the smooth muscle of the body. It excites intestinal peristalsis, causes contraction of the uterus through the sympathetic uterine nerves, and brings about contraction of the bladder with consequent emptying.

As far as the circulatory apparatus is concerned, it causes vasoconstriction and with it, increased blood-pressure. As a cardiac stimulant it acts by slowing and strengthening the heart-beat. The only field of smooth muscle on which

it exerts no vasoconstrictor effect, in fact, the only vessels which it causes to dilate, are those of the kidney, with ensuing diuresis. Intramuscular injections of pituitrin are followed by rise of pressure to 30 mm. Hg.; this rise persisting for thirteen hours.

Klotz[1] found that in animals pituitrin caused a rise in pressure, only when the same was below normal, but it was without effect on normal pressure. Observations on women with uterine hemorrhages resulting in lowered blood-pressure showed the effect of the injection to be most striking.

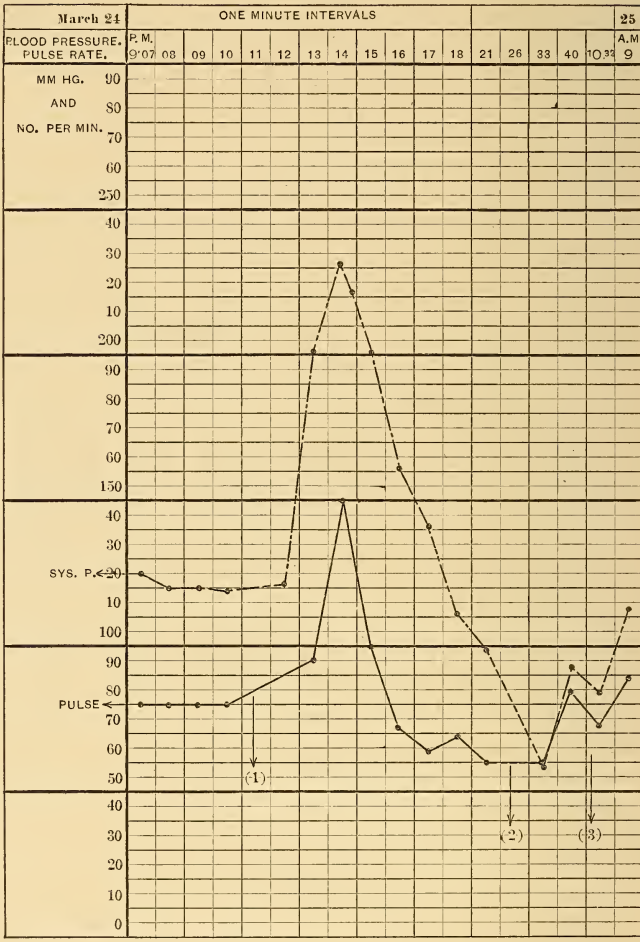
The dose is 0.2 gram fresh glandular substance put up in an ampoule of 1 c.c. (Burroughs, Wellcome & Co.), or in $\frac{1}{2}$ c.c. ampoule with 0.05 gram fresh extract. There seems to be no danger of overdosing, as animals tolerate sixty times this dose. A second injection, before the effects of the first have worn off, has no effect on the pressure. Contra-indications, to its use are nephritis, "goitre heart," arteriosclerosis, and diseased heart muscle. It has been used in shock, and Klotz recommends its administration in pneumonia, diphtheria (?), and diseased heart muscle (?). In man, its administration is without danger, but in animals it causes hepatic and renal degeneration, hypertrophy of the adrenals and heart. It is doubtful if it can produce arteriosclerosis, and it is without organic effect on the heart. I have had no success with pituitrin in cases of persistent hypotension.

10. **Adrenalin.**—The effect of adrenalin is so fugacious that its administration hardly seems feasible. The appended chart (Fig. 57) from Janeway shows how temporary are the effects, and also how alarming may be the depression which follows.

11. **Salt Solution.**—In but one of a hundred cases (other than hemorrhage) have injections of salt solutions produced a rise of blood-presure (Cook and Briggs). When a rise was seen, it was due to purely local and peripheral nervous causes. When the needle is introduced, especially if there is much pain, there is often a momentary rise (10 mm. Hg.), and

[1] Münch. med. Woch., 1911, p. 1119.

FIG. 57



Extreme rise in blood-pressure produced by the intravenous injection of adrenalin. (Janeway's sphygm., 12 cm.) (1) Adrenalin chloride (1 in 1000), ♏xx, injected into the median basalic vein. With the rise, which followed instantly, there was throbbing in head, excitement, substernal pain, and nausea; (2) no pulse palpable; heart sounds very faint; patient delirious, pale. Strychnin sulphate, gr. $\frac{1}{30}$, and atropin sulphate, gr. $\frac{1}{200}$, given hypodermically; (3) caffein sodium salicylate, gr. ii, given hypodermically. In spite of the alarming secondary depression (an unusual result) there was no bad after-effects.

as the solution flows into the tissues, and as the local tension increases, the blood-pressure rises to a considerable degree (20 to 40 mm. Hg.). With this rise the pulse feels stronger and fuller, as if the bloodvessels were filling. The rise in tension is at first rapid, then slower, then begins to fall slowly, the blood-pressure reaching normal in from fifteen minutes to half an hour.

If the infusion be intermittent, instead of continuous, the blood-pressure will be found to rise or fall as the fluid is introduced or interrupted. When giving a continuous infusion of 1000 c.c., lasting an hour, the blood-pressure will return to normal long before the infusion is completed.

The writers believe that any effects from salt solution are due to peripheral reflex action on the vasomotor centre and can be duplicated by any form of intermittent or continued nerve irritation.

CHAPTER XVI.

TREATMENT OF HYPERTENSION AND HYPOTENSION.

Treatment of Hyperpiesis.—In the treatment of hyperpiesis an important fact must not be lost sight of, namely, that high blood-pressure is often a necessary accompaniment of altered function and structure, and were it not for this raised blood-pressure, life could not be carried on with these functional or organic changes. One finds himself between Scylla and Charybdis, between the danger which threatens if pressure remains high and the danger which is certainly imminent if pressure is too suddenly reduced, or reduced too low. For there is a real danger in prolonged hypertension, that of arterial degeneration, hemorrhages, and cardiac failure, and there is the undoubted danger of sudden collapse or of cardiac insufficiency if the pressure is kept too low. It will be recalled that cardiac strength and peripheral resistance are necessary for the maintenance of blood-pressure at a certain level, and it must not be forgotten that the increase of blood-pressure may be the expression of a *rearranged* vasomotor mechanism, which is the only one, under the circumstances, capable of keeping the heart functionating. In individuals with normal blood-pressures, to reduce the pressure below a certain physiological limit is a serious event, as we have seen in previous chapters. What the physiological limit for each individual is, no one can say; it is a variable but never a negligible quantity. Increased blood-pressure has of necessity a raised physiological limit below which blood-pressure cannot be reduced with impunity any more than can blood-pressure in health be reduced below the normal physiological limit.

Some regard hypertension in certain conditions of neph-

ritis as indispensable for the maintenance of function. Krehl believes that the high blood-pressure is but compensatory in order to bring about elimination of toxic products. If so, then limiting the formation of such substances will automatically reduce blood-pressure, as is often observed when a pure milk diet, or salt-poor diet, is employed, but to lower blood-pressure by therapeutic measures, when the intake is furnishing the dangerous metabolites, is to rob Nature of the only means of defense which she now possesses.

When measures are given to combat high blood-pressure their effects should always be controlled by the sphygmomanometer.

The first indication of any rational treatment of hypertension (hyperpiesis) is to ascertain, if possible, the exciting cause or the chief contributing causes, and it is more by the correcting of these than by any drugs that good is accomplished. If business, professional, or domestic cares are at the bottom of the trouble, these should, if possible, be removed, or made to occupy a less prominent place in the patient's life.

We have learned the effect of emotion on blood-pressure, and Cannon and de la Paz have demonstrated clearly that the epinephrin content of the blood is raised over the normal in an angry animal. Can there be an application of this truth to man? The tragic death of John Hunter in St. George's Hospital, following an attack of choler, is too well known to need more than mention, but its lesson is not so well comprehended.

If finances permit, absence from home in a warm, balmy, equable climate should be urged, with congenial surroundings, away from the superheated atmosphere of the modern life. The patient should have rest from mental worries as well as from physical exertion. The latter does not forbid moderate exercise, golf and walking, but aims at the lessening of the terrific pace at which we are living.

Diet.—The reader is referred to the full extract of Hecht's article given in Chapter XV.

Habits.—These should be inquired into most thoroughly. Habits of work and habits of play should be understood

as well as habits of tobacco and alcohol. One can play too hard and one can work too hard, and both should be regulated. Tennis, for instance, is a game for youth, and a man past fifty-five should play it very moderately if at all, and then preferably doubles. Tobacco should be tactfully interdicted, and gradual discontinuance is better than sudden abolition. If the patient has been an excessive smoker, to discontinue the habit abruptly is apt to have more serious consequences than the hypertension, which we are aiming to correct. Nevertheless, tobacco should eventually be absolutely forbidden. The opinions vary as regarding alcohol. Of itself, alcohol has little action on blood-pressure, but secondarily it has a pernicious effect on the bloodvessels, leading to arterial degeneration (arteriosclerosis) and for this reason temperance is advisable. Total abstinence is rarely required, and the advice to abstain is seldom adhered to, for both of which reasons, alcohol may be permitted in great moderation, preferable in the form of whisky. One should not worship at the shrines of Venus, Bacchus, Vulcan and Minerva with too much assiduity.

Elimination.—Elimination should be increased. To stimulate elimination by the kidneys, water is no doubt of great value, but in nephritic hypertension, the advisability of free water drinking is doubtful. It seems as unreasonable to stimulate a diseased organ as to make a broken limb perform its natural function; both demand rest. In cases of hypertensive cardiovascular disease, water is contra-indicated, as liquids tend to raise blood-pressure. If water is to be taken it should be between meals, and the taking of fluid with the meals should be greatly restricted.

Sweat baths, either steam or dry heat, are very useful, but the excessive thirst which sometimes follows, should not be yielded to, and no water should be drunk. Moderate massage following the bath is recommended.

The bowels should be kept loose. It is far better to have free purgation than a condition of constipation. Saline purges taken in hot water before breakfast and a

blue mass pill (0.3 gram) once or twice a week will secure the desired effect.

In robust individuals, venesection is often followed by benefit, but the author has never seen any permanent good result. Cushing's advice regarding venesection in cases of cerebral hemorrhage should be remembered.

High Frequency Currents.—High frequency currents seem to have met with approval in experienced hands.

Drugs.—As far as drugs are concerned, they are of less value than diet, free purgation and hygiene. The fact that hypertension often causes cardiac decompensation and that this cardiac decompensation increases the hypertension, is sufficient indication for the employment of digitalis. High blood-pressure is not a contra-indication to its use, as digitalis, given by mouth, is inert as far as any specific action on pressure is concerned. Indeed digitalis administered to these cases with failing heart may be a means of lowering the hypertension by relieving the cardiac decompensation. Iodids, as stated in previous pages, possess little value unless syphilis is an underlying factor of the high pressure. Sodium nitrite, nitroglycerin and erythrol tetranitrate may be used, but the effects, in the best cases, are transitory (see Chapter XV). If the hypertension is associated with paroxysms of pain, pearls of amyl nitrite (0.12 to 0.3) may be carried on the person and used as occasion demands. I have seen good results follow this prescription.

Potassium nitrate	0.65
Potassium bicarbonate	0.65
Sodium nitrite	0.03–0.13

This is to be taken on rising in 100 c.c. of Apenta water. In cases of extreme hypertension, one may add to the powder 0.015 to 0.13 gram erythrol tetranitrite.

The treatment as outlined above has necessarily been given in general terms and applies to the cases of hyperpiesis without cardiac decompensation. When the heart begins to falter, and signs of the beginning lagging of the right heart make their appearance (see Chapter V), rest

in bed is imperative. The diet which I am fond of prescribing consists of milk, given four times a day, in portions of 200 c.c. No other fluid is allowed. Digitalis is here indicated, and I have seen good results from digipuratum, given in doses of 0.09 gram three times a day. Morphin, when the patient is restless or nervous, is most valuable. Sweat baths frequently repeated, are not contra-indicated, and good results have been seen in those cases which have at first appeared to be too weak to stand them. Active purgation should not be neglected. Venesection is once more being recognized as a useful measure, and in those cases of decompensating hypertensive cardiovascular disease, the removal of 200 c.c. of blood is often most useful.

Prognosis of Hyperpiesis.—In the first phase of hyperpiesis, especially if one is fortunate enough in seeing patients early, the heart and bloodvessels have undergone relatively slight damage, if any, and the prognosis is good if a rational mode of living is followed. When arterial changes are seen, it may be said that the younger the individual, the worse the prognosis, especially when treatment has not proven efficacious. Just how great the arterial changes are, we can surmise only from palpation of a very few vessels, and this, of course, affords but little idea of the degree of sclerosis of hidden vessels, notably those supplying the viscera. A careful eye examination is helpful in forming an opinion, *quoad vitam,* damaged retinal vessels making the outlook much more gloomy than it would be with intact arteries.

Signs of decompensation always render the prognosis guarded (see Chapter V). Apart from the dyspnea of rest, the earliest sign is tachycardia, and the slower the pulse rate, the better the prognosis. A pulse rate above 90 is to be regarded seriously. The pulsus alternans renders the prognosis grave. In cases of hyperpiesis, when blood-pressure begins to fall and the symptoms begin to become prominent, a guarded prognosis should be made. The appearance of some albumin in a large amount of urine of low specific gravity is not of much consequence, but a sudden increase with scanty urine is a danger signal.

Treatment of Hypotension.—The treatment of hypotension depends essentially on the cause of the hypotension. With the exception of epidemic cerebrospinal meningitis, acute infections are hypotensive, but this hypotension, as a rule, calls for no treatment. When the hypotension is causing grave symptoms, rapid weak pulse, embarrassed respiration, and signs of impending collapse, treatment is, of course, indicated. In pneumonia, the falling of blood-pressure below the pulse-rate (see Pneumonia) is a fair index when to begin treatment. Treatment of these emergencies—shock, acute infections, collapse from any cause—is now well understood, but the continued use of the sphygmomanometer makes the indications for treatment more certain.

There is much evidence to show that fatigue and waste products accumulate in a muscle which is doing work or which is tired from much activity. Gruber has lately shown that the height of contraction of a fatigued muscle may be increased 100 to 125 per cent. when the blood-pressure is below 90 to 100 mm. Hg., but only 5 to 25 per cent., when the pressure is above 100 mm. Hg., or in other words, when the blood-pressure is low a small rise has many times the effect that it has when the pressure is high. This, Gruber believes, is due to the increase of efficiency in the circulation following the rise in pressure, thereby carrying away more rapidly the fatigue products which have accumulated in the muscle.

He also demonstrates that lowered arterial pressure is followed by a decrease in the height of muscular contraction. In the experimental animal a fall in pressure from 120 to 100 mm. Hg., was accompanied by no appreciable decrease in the height of contraction; when to 90 mm. Hg., the decrease was 2.4 per cent.; when to 80 mm. Hg., a decrease of 7, and when to 70 mm. Hg., a decrease of 17.3 per cent.

There occurs in practice, with greater frequency than is suspected, a class of cases, which runs a persistent low blood-pressure, pressures below 110 mm. Hg. To these

the author has recently called attention,[1] pointing out a common symptom-complex headache, vertigo, mental and physical tire. There is still another group of cases of hypotension, which is doubtless due to malnutrition, no demonstrable organic disease having been found (tuberculosis, carcinoma, anemia, Addison's disease).

These long-standing cases of hypotension associated with the above-mentioned clinical state show great improvement when the blood-pressure is raised. It is sometimes a difficult task to cause any increase in tension, but it is gratifying to note that even a small rise of pressure is followed by a subjective improvement, the latter out of proportion to the increase in pressure. In the treatment of these cases general hygienic measures are more important than drugs.

Such patients should be instructed to retire at a reasonably early hour, and to rise at a certain fixed time.

Immediately on rising they are to perform a series of exercises, consisting of free movements, somewhat after the following plan.

Daily Exercises.—1. Stand erect with the arms extended, and the fingers in contact above the head. Stoop forward and try to touch the floor with the finger tips, moving slowly, and return again to the original position.

2. Lie at full length on the floor or bed upon the back, with the hands under the hips, and bring each leg alternately and slowly to a position at right angles to the body.

3. Perform the same motions with both legs conjointly. Lying at full length upon the back, the hands clasped behind the head, bring the elbows in, bend the body forward till the face touches the knees, returning again *slowly* to the original position.

4. Standing position, extend the arms sidewise, the elbows straight, until the hands meet above the head. Stretch the arms as much as possible in doing this. Inhale deeply as the arms go up, and exhale slowly as they come down.

[1] Amer. Jour. Med. Sci., 1914, cxlvii, p. 503.

5. With the hands on the hips, the thumbs behind, without moving the feet turn the trunk as far as possible to the right, then to the left. As the body turns rub the fingers deeply into the abdomen.

6. Raise the arms laterally, the palms upward, until the level of the shoulders is reached, bring them forward until the fingers touch; then reverse the movement, extending the arms backward as far as possible, at the same time rising upon the toes.

7. *Breathing.*—Hands at hips, head bent backward, inhale through nose; head erect, exhale through nose and mouth, five to ten times.

Time.—On arising.

Temperature of Room.—As cold as temperature outside.

Windows open.

In order that the exercises should have the desired effect, they must be performed regularly, every morning, at least ten minutes being devoted to the task. They should at first be performed slowly, with intermissions of a few seconds for rest, and deep breathing. As the muscles become accustomed to the work, they may be increased in rapidity and duration, but never to the extent of producing exhaustion or prolonged breathlessness.

Following these exercises, or some similar to these, a shower bath should be taken, first hot, and then as cold as can be well born. If there are no such appointments, a good substitute may be obtained in the following:

Cold Sponge Bath.—To be taken:

1. After morning exercise.
2. On rising.

Stand erect in the tub in which a little hot water remains, and sponge the entire body quickly with cold fresh or salt water; or pour a pitcher of cold water over the back and spine, or use a douche or spray. This should be immediately followed by a brisk rub with a coarse towel, continued long enough to produce redness of the skin. Following this, rest in the recumbent position, well covered up, for five or ten minutes before dressing. Bath room should be warm.

Definite rules should be given for the number of hours' work to be done in each day, and a certain fixed amount of relaxation should be insisted upon. Golf, riding, tennis, walks in the country, are all useful in bringing about a temporary dropping of business, professional and domestic trials. Exercise should be regulated according to the age and physical condition of the subject. A youth with hypotension can be ordered to do much more violent exercise than can a man of middle life, and for the former, for instance, tennis may be recommended, whereas the older man's strength is sufficiently taxed by the more moderate game of golf.

Tincture of nux vomica in ascending doses will be found to be advantageous, beginning with 15 drops three times a day and increasing 3 drops a day. The limit to the increase is seen in the first sign of the physiological action of the drug, and when this is recognized, the single dose is dropped 5 drops and maintained there for a period of two to five days. After this the number of drops is decreased 15 drops a day to the original dose, maintained there for a week, and then discontinued.

Given in this way unusually large doses may be given. Patients not uncommonly have taken 50 drops three times a day, and 75 drops were taken by one of my patients.

Excessive indulgence in tobacco, alcohol, tea, and coffee should be corrected. These and other detrimental habits, too individual to enumerate, should receive due attention, and one should strive by a sensible conduct of life to bring the cardiovascular function to a normal state of activity.

It is very difficult to raise blood-pressure in such cases, but it is encouraging to note that but small rises are associated with considerable improvement.

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